CHAPTER 7

EXPOSURE ASSESSMENT IN PASSIVE SMOKING

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Introduction

There are currently no direct measures of the dose of ETS absorbed in a population under study; however, exposures to ETS can be assessed by personal air contaminant monitoring, modeling of concentrations (based on air sampling, time activity patterns, and questionnaires), or biological markers. (NRC, 1986) Exposure to ETS is determined by the following paradigm: exposure equals the product of respiration rate, concentration, and duration. Concentration is directly proportional to the product of number of smokers, smoking rate, and emissions per tobacco product, and is inversely proportional to the product of space volume and removal rate. The nonsmokers' total exposure to ETS is determined by the product of the time-weighted sum of the concentrations encountered in various microenvironments, and the individual's respiration rate during exposure. (IARC, 1987)

In the epidemiologic studies of passive smoking and lung cancer described under Hazard Assessment, exposures were estimated on the basis of a questionnaire which assesses smoking status, and typically asked simple questions of the sort: "if you are a nonsmoker, do you live with, or work with, or have regular contact with persons who are nonsmokers?" (NRC, 1986) Some studies assessed past exposure history and spouses' smoking rate as well. This kind of question, though useful, is not likely to be fully reliable or precise, particularly for non-domestic exposures. (NRC, 1986; IARC, 1987) On the other hand, it has been shown that those nonsmokers who report exposure to ETS at home tend to have higher non-domestic exposures as well.(NRC, 1986; Wald, 1986)

Ideally, the health effects of ETS exposures might be assessed by quantifying the time-dependent exposures and doses for each of the several thousand compounds in tobacco smoke and defining dose-response relationships for these compounds in producing disease, both as isolated compounds and in various combinations. However, the enormity of this task has led to simpler approaches which attempt to use measures of exposure to individual smoke constituents as estimates of whole smoke exposure. For this reason, exposures to ETS are often assessed using markers of the

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vapor phase or particulate phase. Although biological markers show promise as measures of exposure, they also have limitations. Another consideration is averaging time. For chronic diseases such as cancer, average exposures occuring over a year or lifetime are of greater importance than short-term exposures.(SG, 1986)

The two most promising markers for ETS are respirable suspended particles in the size range ≤2.5 um (RSP) and nicotine and its metabolite cotinine.(NRC,1986; SG,1986; IARC,1987) A majority of field studies have used RSP as an indicator of exposure to ETS because of the substantial emission of RSP in indoor spaces from tobacco combustion. ETS is the dominant contributor to the indoor levels of RSP. The total RSP, as measured by personal monitors, has been found to be substantially elevated for those who report exposure to ETS relative to those who report no exposure. Both air monitoring and modeling clearly indicate that RSP concentrations will be elevated over background levels in indoor spaces when even low smoking rates occur.(NRC, 1986) Although lacking specificity for tobacco smoke, the prevalence and number of smokers correlates well with RSP levels in homes and other enclosed areas.(SG,1986) RSP is the single largest component of ETS by weight, and RSP is currently the best and most-utilized general category of air contaminants to represent ETS.(NRC,1986)

The biological markers that have been most useful for assessing recent exposures to ETS are nicotine and its metabolite cotinine, which derive exclusively from tobacco products, of which tobacco smoke is the most important source. Almost all nicotine shifts from the particulate phase in MS and fresh SS smoke to the vapor phase in ETS. Nicotine and cotinine can be quantified in saliva, blood, and urine. Generally, the mean concentrations of nicotine and cotinine in plasma or urine of nonsmokers exposed to ETS under natural conditions is about 1 percent of the mean values in smokers, (NRC,1986) reflecting the fact that smokers are present in nearly all environments, including most workplaces, restaurants, and transit vehicles, making it almost impossible for nonsmokers to avoid some exposure to ETS.(SG,1986)

A. Sources of ETS

In 1986, an estimated 50,000,000 US smokers aged ≥17 yrs smoked about 584 billion cigarettes annually. (NRC, 1986; Tobacco Institute, 1987) They consumed an additional 3.2 billion cigars, as well as an estimated 24.4 million pounds of tobacco for pipes and hand-rolled cigarettes.(NRC, 1986) The average US cigarette smoker smokes 32 cigarettes per day at a rate of 2 cigarettes per hour and emits about 22 mg of RSP per cigarette.(IARC, 1987) Since the average person spends about 90% of the time indoors, an estimated 12,000 metric tons of RSP are emitted into US indoor

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microenvironments each year from cigarette smoking alone. Assuming cigars produce 3 times as much RSP as cigarettes and that pipes produce as much RSP as a cigarette, (Repace and Lowrey, 1982) where pipes and hand-rolled cigarettes are assumed to contain 1 g of tobacco, then all cigars are estimated to contribute as much RSP indoors as 11 billion cigarettes, while all pipes and hand-rolled cigarettes are estimated to contribute as much as 15 billion regular cigarettes, increasing the estimated total RSP generated in US indoor microenvironments from all cigarettes, pipes, and cigars to nearly 13,000 metric tons per year. As exemplified by data from EPA's TEAM study, ETS predominates over other sources of RSP indoors (see Fig. 3.)

Although the percentage of the population that smokes has declined from nearly 50% in the 1960's to about 30% presently (OSH,1988), the percent of smokers who are heavy smokers has increased steadily over the past 30 years; thus although the percentage of smokers has gone down, the increase in smoking rate may tend to offset that trend towards lowering nonsmoker exposure to ETS.(NRC,1986).

B. Indoor air transport of ETS

Nonsmokers are exposed to ETS in indoor spaces. The determinants of these enclosed-space exposures include smoking occupancy, source air-contaminant emission characteristics, source use, building characteristics, space volume, infiltration or ventilation rates, efficiency of air mixing, surface sorption, chemical transformation, and the efficiency of air cleaning equipment. The interaction of these variables in determining the resultant concentrations of ETS has been evaluated in both controlled laboratory settings and in field studies within the theoretical framework of the mass-balance equation. The mass-balance equation may be applied to tobacco smoke either as an equilibrium model (time-independent) or as a dynamic model (time-dependent). Dynamic and equilibrium models are useful in laboratory studies; equilibrium models are best suited to evaluating and predicting ETS concentrations in field studies, particularly when average concentrations over a period of days or longer are of interest. (NRC,1986)

Laboratory and field studies typically utilize some form of a single-compartment equilibrium model to evaluate the input parameters of the mass-balance equation, to evaluate field study data, and to project RSP concentrations from ETS indoors. These studies have reduced the general single-compartment mass-balance equation to the following simplified form:

$$C_{eq} = G[m(N_v + N_s)V]^{-1} \qquad (1),$$

where C_{eq} is the equilibrium concentration of ETS-generated RSP in a space, expressed in units of micrograms per cubic meter

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(ug/m^3), G is the RSP generation rate from tobacco combustion in units of micrograms per hour (ug/hr), N_v is the ventilation or infiltration rate in units of airchanges per hour (ach), N_s is the loss rate of RSP due to surface removal in a space in air changes per hour, V is the volume of the space in cubic meters (m_3), and m is the mixing rate expressed as a fraction. The above model assumes no air-cleaning devices, either in the space or recirculated air; Leaderer (1984) has given a detailed review of this model. Under laboratory conditions, these input parameters can be controlled and evaluated. In conducting field studies or in estimating past RSP levels indoors, the values on the right side of eq.1 have to be determined from available data. This equation assumes equilbrium conditions, and to the extent that any of the generation or removal terms are intermittent (e.g. smoking rate) or variable (e.g. ventilation rate), errors are introduced. (NRC,1986)

The most extensive use of the mass-balance equation for assessing RSP levels due to ETS in occupied spaces has been due to Repace and Lowrey (1980). (NRC,1986) Drawing upon the "best available data" from several sources, including both measured and estimated parameters, they proposed and applied in field observations a condensed version of the mass-balance equation for estimating RSP exposures due to ETS in a variety of indoor microenvironments.(NRC,1986) Their model is:

$$C_{eq} = 650\ D_s/N_v \qquad \{2\},$$

where C_{eq} is the equilibrium concentration of RSP due to ETS in units of micrograms per cubic meter, D_s is the density of active smokers (burning cigarettes) observed in a space per 100 m^3 over the sampling time, and N_v is the ventilation or infiltration rate in ach.(NRC,1986) The constant term (650) is calculated from a standard set of assumed conditions for smoking rates, RSP emission rates, mixing factors, ventilation rates, and sink rates. These standard sets of conditions are derived largely from experimental data and building standards.(NRC,1986) In applying equilbrium mass balance models such as eq.{2}, gathering data on easily measured input parameters such as smoking rates or volume can substantially reduce the variablity of the estimated RSP levels. However, the NRC stated that additional field testing of eq.{2} as well as a better understanding of the variability of the input parameters was needed.(NRC,1986)

More recently, the International Agency for Research on Cancer (IARC) has published derivatives of eq.2 which incorporate advances in understanding.(IARC,1987) Eq.{2} assumes a steady generation of tobacco smoke, which is generally only valid when 3 or more smokers are present in a space. It is also limited for modeling purposes by being based on the room density of active smokers. The derivative equations incorporate adjustments which allow for intermittent generation of smoke when less than 3

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smokers are present, and also are based on the room density of habitual smokers (number of habitual smokers per 100 cubic meters). Thus, the presence of a typical or "habitual" smoker (i.e., one who is assumed to smoke at an average rate of 2 cigarettes per hour at 10 minutes per cigarette, with an emission factor of about 22 mg of SS RSP per cigarette) is the modeling parameter rather than the room density of burning cigarettes.(IARC, 1987) These derivative equations are given as follows:

$$C_{eq} = 217\ D_{hs}/N_v \qquad (3),$$

where C_{eq} is the equilibrium concentration in units of micrograms per cubic meter, D_{hs} is the number of habitual smokers per 100 cubic meters of space volume, and N_v is the number of space air changes per hour. Eq. 3 assumes that the number of habitual smokers in the space is 3 or more, so that the generation of smoke is steady. If the number of habitual smokers being modeled is only 2, then eq. (4) applies, yielding:

$$C_{eq} = 145\ D_{hs}/N_v \qquad (4),$$

for the 2-smoker equation, where the smaller coefficient approximates the effect of the non-steady generation of ETS. Similarly, if the number of habitual smokers to be modeled is one, then eq.(5) is used:

$$C_{eq} = 72\ D_{hs}/N_v \qquad (5),$$

for the single-smoker equation. The use of such equations is illustrated as follows: As part of the the Harvard 6-City study of indoor and outdoor air quality, Spengler and colleagues (1981) collected RSP samples in 55 homes in 6 cities between May 1977 and April 1978. The number of smokers living in each home was recorded. The quantity of tobacco smoked was not reported, nor was the number of hours each smoker spent in the home, and the air exchange rates were not measured. The average "background," or mean indoor RSP level in the homes of nonsmokers was about 24 ug/m^3; using regression analysis, the authors estimated that the average impact of a single smoker (a composite averaged over both sexes) on 24-hr average RSP levels in a residence was about 20 ug/m^3. On average, two such habitual smokers would make about 40 ug/m^3 (24-hrs); allowing for 8 hours of sleep, the 16-hr average would be 60 ug/m^3.

Although the air exchange rates were unknown, Fig. 4 shows a histogram of the frequency of occurrence of various air exhange rates (called infiltration rates) during the heating season for typical middle income housing (R. Grot, personal communication) for 266 homes in 14 cities around the US in 1978 (SG,1986). Occupants were asked to keep windows and doors closed during the tests. Under these conditions, the mean air exchange rate found

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was 1.1 ± 0.9 ach (Grot & Clark, 1979); this value is likely to be lower than a full seasonal average with no restrictions on door and window openings. Based on time-budget studies, in a two-smoker home, on average, an estimated 21 cigarettes per day (cpd) would be smoked in the home by the homemaker, and 11 cpd by the wage-earner, for a total of 32 cpd, by a smoking couple (i.e., assuming 2 cigarettes per hour, this implies that smoking occurs in the home 16 hrs out of 24).(Repace and Lowrey, 1985)

The utility of Eq. 4 depends on the assumption of an air exchange rate. Eq. 4 for a two-smoker home (D_{hs} = 0.58) predicts a value of C_{eq} = 60 ug/m^3 16 hr average or 40 ug/m^3 24-hr average, for a value of N_v = 1.4 ach. Adding the 24 ug/m_3 background yields an estimated 64 ug/m^3 for a 24-hr average. Extrapolating from this to a single smoker home would place the estimated 24-hr RSP concentration at 44 ug/m_3, and to a 3 smoker home yields 84 ug/m^3. This is consistent with observations as shown in fig.5. Thus, by assuming an air exchange rate only a third of a standard deviation from the 14 city mean, the 6-city RSP levels in smokers' homes can be fit exactly. If the 14 city mean of 1.1 ach were used for N_v instead, the predicted concentrations would still be within 27% of observations. This example illustrates the utility of models in estimating nonsmokers' exposures to ETS.

As a second example, consider the measured aerosol mass concentration in a 700 m^3 (25000 ft^2 floor area) office with one smoker (smoking rate not reported), and a measured air exchange rate of 1 ach (see Fig. 6); the large impact on the office aerosol concentration caused by smoking is apparent by comparing the daytime and evening RSP concentrations.(IARC, 1987) The predictions of eq. 5 for D_{hs} = 1 smoker (smoking at a rate of 2 cph) per 7 hundred cubic meters and N_v = 1 ach yields C_{eq} = 10 ug/m^3; with an 18 ug/m^3 background added, the predicted RSP level is 28 ug/m^3. The predicted value (eq. 3) for chainsmoking (6 cph) is 48 ug/m^3, which is consistent with the daytime data in Fig. 6. It is clear from fig. 6 and also from models, that ETS can be very persistent in indoor environments: at an air exchange rate of 1 ach, it takes 3 hrs for 95% of the smoke from 1 cigarette to be removed (IARC, 1987)

Recent research has revealed several interesting factors in large office building air exhange. There are many pathways for floor-to-floor air communication, particularly return air shafts, where the existence of such pathways can cause a building's air exchange characteristics to closely approximate those of a single large open space; it does not require unusual numbers or sizes of openings to create these conditions.(A.Persily, personal communication; Persily and Grot,1986) This implies that ETS may diffuse throughout a large office building, exposing nonsmokers even in private offices. Nicotine measurements in office buildings support this observation. (Williams, et al., 1985) A sampling of whole-building air-exchange rates in 8 large federal

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office buildings in 7 states with different climates is shown in Fig. 7, and these generally approximate the ASHRAE 62-81 ventilation standards for offices (20 cfm per occupant, equivalent to 0.84 ach, for smoking buildings), on average, although there are some buildings significantly lower. A recent EPA study of air exchange rates in 6 buildings (3 new, 3 old) did not show significant differences between the new (.5 ach) and old (.5 ach) buildings' airchange rates, although for a given building nighttime measurements tended to be lower. (Sheldon, et al.,1987)

In summary, limited field tests of the general equilibrium model, in which some of the input parameters are measured and others are estimated from either chamber studies or building codes, have predicted RSP levels reasonably well over a wide range of values of input parameters. It is clear that both models and observations based on personal monitoring or area monitors in various microenvironments yield consistent results: RSP levels when smoking is allowed will result in substantial increases over RSP levels in nonsmoking occupancy.(NRC,1986)

C. Measured concentrations of RSP from ETS

Both chamber and field studies (Table 8) have demonstrated that tobacco combustion has a major impact on the mass of suspended particulate matter in occupied spaces in the size range <2.5 um, defined here as RSP. RSP is a major component of ETS. Even under conditions of low smoking rates, easily measurable increases in RSP have been recorded above background levels (Table 8). The term RSP, however, encompasses a broad range of particulates of varying chemical composition and size emanating from a number of sources (outdoors, cooking indoors, kerosene heaters, etc.)(NRC, 1986) The apportionment of RSP indoors depends primarily on the presence of these other sources, however, there are few indoor sources generating concentations which approach in strength those due to ETS. As section II indicates, there appears to be little variability between brands of cigarettes or tobaccos for RSP emissions, although cigars will produce greater emissions than cigarettes. Thus, it may be inferred from Table 8 that from a comparison of smoking and nonsmoking buildings, the bulk of the RSP found in buildings where there is smoking is due to ETS. For example, by comparison of the data of First (1984), Leaderer, et al (1986) and Repace and Lowrey (1980,1982) for a total of 42 smoking buildings and 21 nonsmoking buildings, the weighted average RSP level in the smoking buildings is 262 ug/m^3, while in the nonsmoking buildings it is 36 ug/m^3, suggesting that about 85% of the indoor RSP levels in those buildings is due to ETS. Most of the buildings involved were public access buildings.

Figure 8, (IARC, 1987) a plot of the data of Repace and Lowrey (1980, 1982) illustrates the large impact of smoking on RSP

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levels; smoking data points 'A' thru 'T' encompassed a wide variety building microenvironments,including 10 restaurants, 3 cocktail lounges, 3 bingo games, 2 dinner-dance halls, 1 bowling alley, 1 sports arena, 1 hospital waiting room, and a residence during a dinner party. Studies of the dispersion of RSP from ETS in US homes showed at most a factor of 2 difference among various rooms in residences, averaged over 24 hrs. In a setting such as a work environment, where the average exposure is several hours, ETS would be expected to disseminate throughout the airspace where smoking is occuring.(SG, 1986) Although most people spend approximately 90 percent of their time in just two microenvironments (home and work), important exposures can also be encountered in other microenvironents, e.g., in transit, which accounts for 0.5 to 1.5 hrs per day for most people..(SG, 1986) Exposures on aircraft can also be considerable. (Repace and Lowrey, 1988)

D. Exposure of nonsmoking populations to ETS

In the general population (both sexes) aged $\geq$ 17 years in 1980 (160,798,000 persons), a majority has smoked at some time: 32.6% were current smokers, and 21.3% were exsmokers, while 46.1% had never smoked (see Table 9). Among current 1980 smokers, 53% were male, and 47% were female, with some race- and gender-specific differences : white males, 35.9%, black males, 42.0%; white females, 29.3%, black females, 29.7%. (R. Wilson, NCHS, personal communication) In terms of the population at risk, both lifelong nonsmokers and former smokers, 66.7% of the adult population and the overwhelming majority of children are potentially at risk from involuntary exposure to ETS (in 1970, Bonham and Wilson (1970) found in a national probability sample of children, that 62% of US homes with children contained 1 or more smokers). Exposure to various subpopulations or individuals, however, may vary considerably. For example, the prevalence of smoking among subgroups of the population who proscribe smoking on religious grounds (such as Mormons and Seventh-Day Adventists) is much lower: for example, in 1980, only 1.7% of Seventh Day Adventist men and 0.5% of Seventh Day Adventist women reported current smoking, although 35% of the total were exsmokers. The incidence of lung cancer -- a disease for which the majority of cases occur in smokers -- among SDAs is 21% of that in the general population. Thus, SDA homes would be, in general, expected to be ETS-free.

The microenvironments of importance for exposure to ETS will be those where the population spends the bulk of its time. As Table 10 (Ott, 1981)(based on 1972 data) shows, employed men spend an estimated 56% of their time at home, and 28% of their time at work, for a total of 84% of the time at home and at work; employed women spend 64% of their time at home, and 22% of the time at work, for total of 86% of the time at home and at work; while homemakers spend 85% of the time at home. When time spent

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in other peoples' homes and in non-work places of business are added in, the population averages about 88% of its time in homes and workplaces. These sites, therefore, must, on average, predominate as potential sites for exposure to ETS for the general nonsmoking population. A UK study of exposure to ETS in 20 nonsmoking men whose wives smoked showed that 78% of the men's reported hours exposure came from outside the home; by contrast, 90% of the ETS exposure of 101 nonsmoking men whose wives did not smoke was reported to come from non-domestic microenvironments. (Table 11). Since the second largest source of time spent by men is in the workplace (Table 10), this suggests the workplace may be an important source of exposure for nonsmoking men.

One recent questionnaire survey of exposure to ETS in a California population subscribing to a health-maintainance plan indicated that 63% of nonsmokers surveyed reported exposures to tobacco smoke (Friedman, 1983); this occurred despite the fact that in the 1980's California has has been in the forefront of restrictions on smoking in public, with 44% of its population currently living in communities that have enacted workplace smoking restrictions.(SG, 1986) Garfinkel (1981), in a study of 176,000 nonsmoking US women (1960-1972), found 72% had smoking husbands. Kabat and Wynder (1986), in a recent study of 215 sixty-year-old US women nonsmokers, found that 65% were exposed at home and 67% reported exposure at work, averaged over adulthood. Studies of the concentration of nicotine and cotinine in the body fluids of nonsmokers report similar results (Table 12); Fig. 10 (Jarvis & Russell, 1984) shows that in a study of about 100 UK nonsmokers, only 12% of the subjects had undetectable cotinine levels. Moreover, in the latter study, surprisingly, nearly 50% reported no exposure, suggesting that ETS permeates indoor atmospheres to such an extent that many nonsmokers are unwittingly exposed. This is borne out by a study of 46 US infants, 40% of whom were reported by their mothers to be unexposed to ETS, but only 20% had undetectable urinary cotinine levels (Fig. 11).(Greenberg, 1986) In a third UK study (Wald, 1986) of urinary cotinine in 221 nonsmokers, the 20% who reported no exposure had mean urinary cotinine levels which were 21% of the remainder of the group who reported exposure.

The foregoing illustrates that exposure to ETS is very widespread in the population, even among those nonsmokers who believe themselves to be unexposed, however it tends to be greater in those who say they are exposed at home, possibly indicating a greater tolerance for ETS among men with nonsmoking spouses. Although there have been numerous measurements of ETS concentrations in various indoor settings, these data do not represent a comprehensive description of the actual distribution of ETS exposures in the US population. Spengler et al.(1985) and Sexton et al. (1984) demonstrated by personal monitoring of RSP and the use of time-activity questionnaires that exposures to ETS both at home and at work are significant contributors to personal

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exposures. However, additional data on the distribution of smokers in the nonsmokers' environment as well as the distribution of ETS levels in that environment, are needed in order to characterize the actual ETS exposure of the population. In the absence of such data, population exposures can be estimated by models or by extrapolation from biological markers from existing studies. (SG, 1986; IARC, 1987)

In summary, exposures to ETS can be assessed by personal air contaminant monitoring, modeling of concentrations based upon air sampling, time-activity patterns, and questionnaires, or upon biological markers. The two best methods at present are based upon the biological markers, nicotine and its metabolite, cotinine, which are present in the saliva, plasma, and urine of active and passive smokers, and upon RSP from the particulate phase of ETS, which has been used by a majority of field studies because of the substantial emission of RSP from tobacco combustion. In US, ETS is generated by 50 million smokers, who smoke the equivalent (including pipes and cigars) of 610 billion cigarettes annually. Although the number of smokers has been declining, the percentage of heavy smokers has been increasing. There are models in use, based on the mass-balance equation, and validated both under laboratory and limited field conditions, which can predict the concentrations of RSP from ETS to a reasonable degree of accuracy. Application of such models, together with field studies of RSP concentrations and behavioral studies, has shown that exposure to ETS is very widespread in the population. Field studies of RSP in buildings where smoking occurs suggest that RSP from ETS contributes 80 to 90 percent of the particulate load during the period of smoking, and that it persists for long periods after smoking ends at typical building air exchange rates, thus prolonging nonsmokers' exposures.

E. Integrated exposure analysis

Exposure to ETS can be quantified either by atmospheric or biological markers. Of the latter, expired carbon monoxide, carboxyhemoglobin, plasma thiocyanate, plasma, urinary or salivary nicotine, and plasma, urinary, or salivary cotinine have been used to evaluate exposure to ETS. However, successful attempts to quantify the degree of exposure have been limited largely to measurements of nicotine and cotinine. Urinary nicotine is a sensitive indicator of recent ETS exposure, while cotinine appears to be the short-term marker of choice for epidemiologic studies. Nicotine and cotinine are the best markers currently available. Levels in body fluids may be elevated 10 or more times in the most heavily exposed groups compared with the least exposed groups. Mean levels of urinary nicotine and cotinine in body fluids increase with an increasing self-reported ETS exposure and with an increasing number of cigarettes smoked per day by active smokers. (SG, 1986)

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Nicotine is found in measurable concentrations in the saliva and urine of most urban nonsmokers, and is present in higher concentrations in those with some recent exposure. Estimating the magnitude of the passive smoking dose is difficult, and it is of doubtful validity to extrapolate from the uptake of one marker to another. Over a period when one cigarette equivalent of carbon monoxide is absorbed, the dose of nicotine appears to be only between 1/10 and 1/3 of a cigarette equivalent. Similarly, under extreme conditions of indoor pollution, it has been calculated that a nonsmoker would inhale volatile nitrosamines equivalent to 10 nonfilter cigarettes or 35 filter cigarettes.(IARC, 1987)

The average concentration of cotinine in the blood of habitual smokers is about 300 ng/ml, and is calculated to represent the consumption of about 36 mg of nicotine per day. On this basis, and on the assumption that formation of cotinine from nicotine and clearance from the body does not differ substantially from smokers to nonsmokers, present data suggest that average urban nonsmokers (in the UK) take in 0.2 mg of nicotine per day.(IARC, 1987) [.2 mg represents .6% of the smokers' dose] The highest plasma cotinine concentration observed in a nonsmoker corresponds to an approximate maximum dose of 2.5 mg of nicotine per day, 10 times higher, and 7% of the average smoker's dose. Recent studies of salivary cotinine in schoolchildren in the UK showed, in the case where both parents smoked, average concentrations just over 1% of the levels seen in heavy cigarette smokers.(IARC, 1987) There is little data on nicotine levels in nonsmoking US adults. However, one recent study of personal exposures to airborne nicotine in 4 US office workers showed about a .1 mg mean exposure (mean personal nicotine concentration of 15 $\pm$ 9 ug/m^3, daily workday average, times a 0.8 m^3/hr inhalation rate times an 8-hr workday). The nonsmokers were exposed to the smoke of a co-worker who smoked 9 cigarettes per workshift, about half the rate of the average US smoker. (Hammond et al.,1987) This result, although limited, is consistent with the UK data. [N.B.: At an RSP/nicotine ratio of 13:1, (Hammond et al., 1987) the associated 8-hr av. RSP level from ETS in this office is calculated at 195 ug/m^3.]

Although the ratio of nicotine to other tobacco smoke constituents differs in MS and SS smoke, nicotine uptake may still be a valid marker for total ETS exposure. Nicotine uptake in nonsmokers has been estimated in terms of cigarette equivalents from various studies to vary between 1/6 to 1/3 of a cigarette per day.

The NRC reports various estimates of cigarette equivalents based upon cotinine in nonsmokers ranging from 0.1 to 1 cigarette per day, and utilizes a ratio of urinary cotinine in ETS-exposed nonsmokers (25.2 ng/ml) to that in active smokers (1826 ng/ml) yielding a 1.4% result.(NRC, 1986) [Assuming a usage of 32

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cigarettes per day by habitual smokers based upon cigarette sales (Repace and Lowrey, 1980), this yields 57 ng/ml/cigarette, or 0.44 cigarette equivalents per day. Adjusting this by multiplying by the ratio of cotinine clearance in nonsmokers to that in smokers reported in one study (Fig.1), 49.7 hrs/18.5 hrs = 2.67, yields a higher value, 1.1 cigarette equivalents per day or 3.6% of the smokers' dose.] In summary, based upon the limited studies (none of which are a probability sample of US nonsmokers) of cotinine in body fluids of nonsmokers (see Table 11, SG, 1986), nonsmokers appear to have of the order of 1% of the nicotine uptake of smokers. However, these estimates must be interpreted with caution; relative absorption of nicotine in smokers and nonsmokers may substantially underestimate exposure to other components of ETS. (SG,1986)

Alternatively, human exposure to ETS can be estimated using approaches similar to those used for other airborne pollutants. Measures of exposure to individual atmospheric smoke constituents can be used as estimates of whole smoke exposure. The accuracy of this approach is limited by changes in the composition of ETS with time and conditions of exposure. One widely reported marker of ETS is respirable suspended particulate matter (RSP). Although lacking specificity for tobacco smoke, the prevalence and number of smokers correlates well with RSP levels in homes and other enclosed areas. In the Harvard study of indoor air pollution in 6 cities, Spengler et al. and Sexton et al. demonstrated by the personal monitoring of RSP and the use of time-activity questionnaires that exposures to ETS at home and at work are significant contributors to personal exposures. In general, measurements in a large number of locations using measures of smoke generation such as the number of people smoking or the number of cigarettes being smoked have shown a definite relationship of smoke generation to particulate levels. In US homes, there are few other sources of RSP, and therefore, the relationships of RSP measurements to ETS are quite accurate.(SG, 1986)

Repace and Lowrey (1980) measured RSP concentration using a piezobalance in several public and private locations, in both the presence and absence of smoking. They then developed an empirical model utilizing the mass balance equation (Eq.2). Using both measured and estimated parameters as input to the model, they validated the model for predicting an individual's exposure to ETS.(SG, 1986) Kuller et al (1986) in a review of estimates of the nonsmoking population's exposure to ETS, observed that cigarette smoking is probably the single most important source of indoor RSP; that a higher percentage of nonsmokers appear to be exposed out of the home, usually at work (Friedman, 1983); and that modeling of RSP has estimated that the average exposure of the nonsmoking adult population to tars from ETS was 1.43 mg/day, varying from 0 to 14 mg (Repace and Lowrey, 1985). Kuller et al. (1986) in reviewing the latter estimate, observed that the ratio

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of average exposure in passive smoking to that in active smoking, was about 0.3%. This translates into 3% of the smoker' exposure for the most-exposed passive smokers, reasonably consistent with estimates based on doses from nicotine and cotinine, above.

Table 13 gives estimates of the probability-weighted exposures to ETS for US nonsmoking adults at home and at work, the two most-frequented microevironments.(Repace and Lowrey, 1985) Table 13 is derived from RSP concentration modeling based upon Eq.'s 2-5, and from assessments of exposure probability based on a limited national survey of top management and health officials concerning prevalence of smoking in the workplace in 3000 US corporations, large, medium, and small (29% response), and a national probability sample of the prevalence of smoking in homes with children (used as a surrogate for all homes). Exposure probabilities were a weighted average taken over the number of workers in white-collar and blue-collar occupations, and including the different exposure probabilities for white and blue collar workers. Air exchange rates and building occupancies were taken from ASHRAE Standard ventilation rate tables for white-collar workplaces (which were used as surrogates for blue-collar workplaces).

Table 13 estimates average the workplace ETS exposure probability at 63%, and the average estimated domestic ETS exposure probability at 62%, where the focus was on estimation of ETS exposures in the 1950's to mid-1970's, since these exposures were held to be of primary significance for the studies of passive smoking and lung cancer, given the long latency for lung cancer. Comparison of these exposure probability estimates to adult life ETS exposure histories taken by Kabat and Wynder (1986) for 215 60-yr old female nonsmokers, 65% at home and 67% at work, shows good agreement. Table 13 estimates a 0.45 mg/day RSP exposure for nonsmokers at home, (weighted for male and female time-activity pattern differences, and for respiration rate) corresponding to a 19 ug/m^3 24-hr average, in good agreement with results (19 ug/m^3 per smoker) published in the 6-City study.(Repace and Lowrey, 1985) Table 13 also estimates a 1.82 mg/day RSP exposure for workers, (again weighted for male-female time-activity patterns and for respiration rate) corresponding to about a 230 ug/m^3 workplace concentration, using ASHRAE Standard 62-73 for workplace occupancy and performing a weighted average workday for the different hours worked by men and women (Repace and Lowrey, 1985). This is in good agreement with the weighted average concentrations (262 ug/m^3) reported for ETS in public access buildings (See section III-C).

In summary, using either the best biological marker (cotinine) or the best atmospheric marker (RSP) produces a consistent assessment of ETS exposure, i.e., of the order of 1% of that in smokers. The most-exposed individuals appear to have levels about ten times higher. Based upon limited data, the typical

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nonsmoker appears to carry a daily body burden of about 0.2 milligrams (mg) of nicotine. The cotinine-based estimates have the advantage that they reflect actual dose of an ETS constituent. They have the disadvantage that they do not reflect a wide distribution of target populations, are based mostly on UK ETS exposures, and may substantially underestimate exposures to other constituents of ETS. The RSP-based estimates have the advantage that they are model-based, can be used to estimate exposures in a variety of microenvironments, represent the great bulk of ETS carcinogens, and can be compared with atmospheric measurements of RSP. They have the disadvantage that they do not represent whole smoke exposure, and do not reflect absorbed dose. The greatest source of uncertainty is that neither cotinine nor RSP measurements are based on a national probability sample, and on an absolute scale, represent a limited amount of data. Nevertheless, the NRC(1986), the SG(1986), and IARC(1987) have utilized this data base for exposure assessment purposes. Estimates of the adult nonsmoking population's exposure to RSP from ETS suggest that the range of exposure is from 0 to 14 mg per day, with the population average put at 1.5 mg per day, where the peak-to-mean ratio is about a factor of 10, consistent with the biomarker-based findings.

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FIGURES AND TABLES FOR CHAPTER 7

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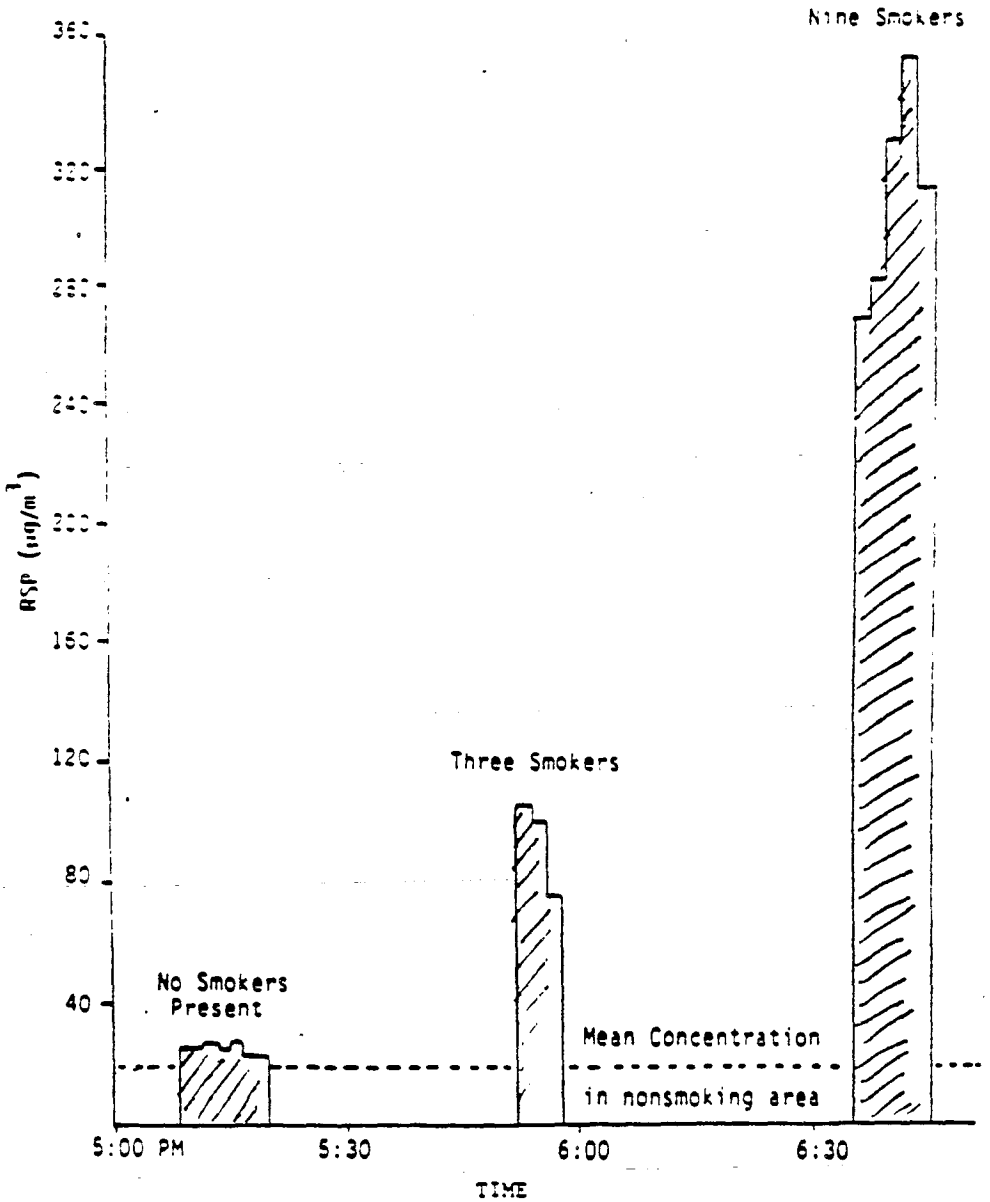
FIGURE 3. EPA's TEAM Study demonstrates that smoking provides the dominant source of RSP in many buildings (Sheldon, et al, 1986)



Respirable particulates in 3rd floor lounge of the elderly home-1 (3/24/83).

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FIGURE 4. Air exchange rates in homes are one determinant of nonsmokers' exposures to ETS. Low air exchange rates mean higher exposures. Shown is a histogram of infiltration values in a sample of 266 older US middle class homes around the country. Average heating season values are shown. The median of the distribution is 0.9 ach and the mean is 1.1 ± 0.9 ach.(Grot and Clark, 1979)(NRC, 1986)

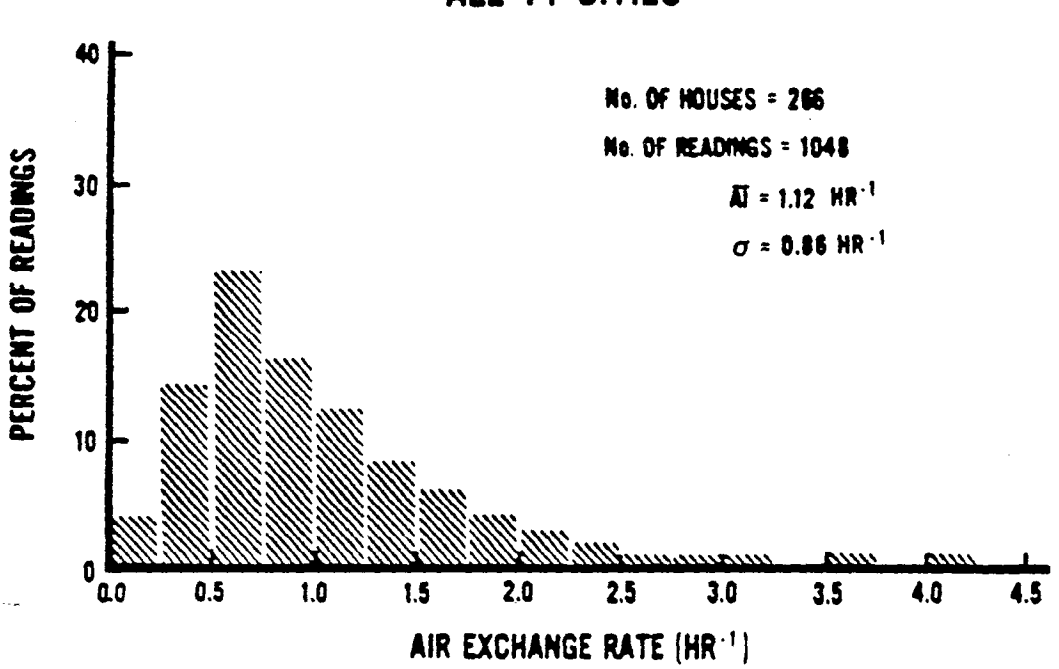

Figure 3. Histogram of measured natural air infiltration rates for 14 weatherization sites

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FIGURE 5. Application of IARC model (IARC, 1987) vs. observations for monthly mean RSP concentrations in six US cities (Spengler, et al., 1981; NRC, 1986) The effect of smoking is to increase backgound residential RSP levels (24 ug/m^3) an average of about 20 ug/m^3. The theoretical values from eq.4 are shown (horizontal solid lines) for comparison, estimated for 1,2, and 3 smoker homes by assuming a mean air exchange rate of 1.4 ach.

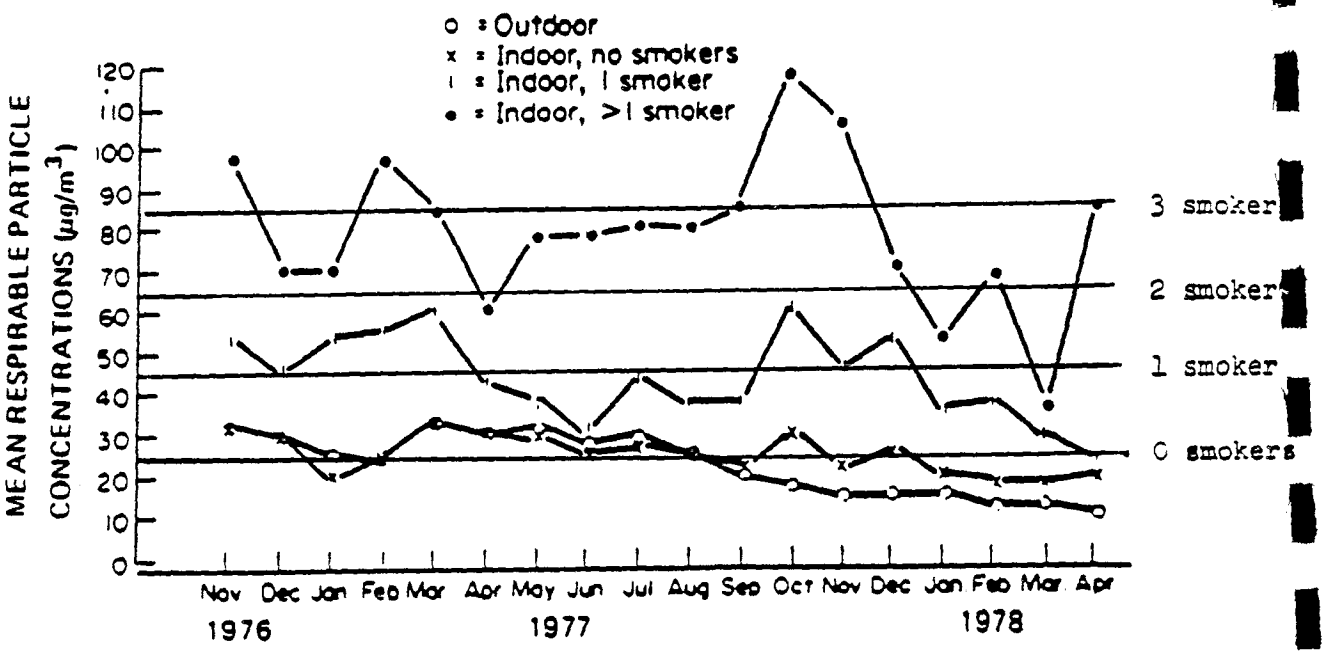

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FIGURE 6. The effect of smoking on workplace RSP levels in a Minneapolis office building. The contrast between daytime RSP levels, when smoking occured, and night-time RSP levels, when it did not, is marked.

Aerosol mass concentration in a 700-m^3 office with one smoker (Nelson *et al.* 1982) ...e smoking instrument and smoking rate were not specified. However, the air exchange rate for the space may be calculated by means of equation 2. For the decay of ETS on Thursday, July 9, a non-linear regression analysis of the RSP levels, with an 18 $\mu g/m^3$ background level subtraction, yields $C_a = 1.0$ ach ($r^2 = 0.95$). This value is close to the ASHRAE-recommended ventilation rate for office space.

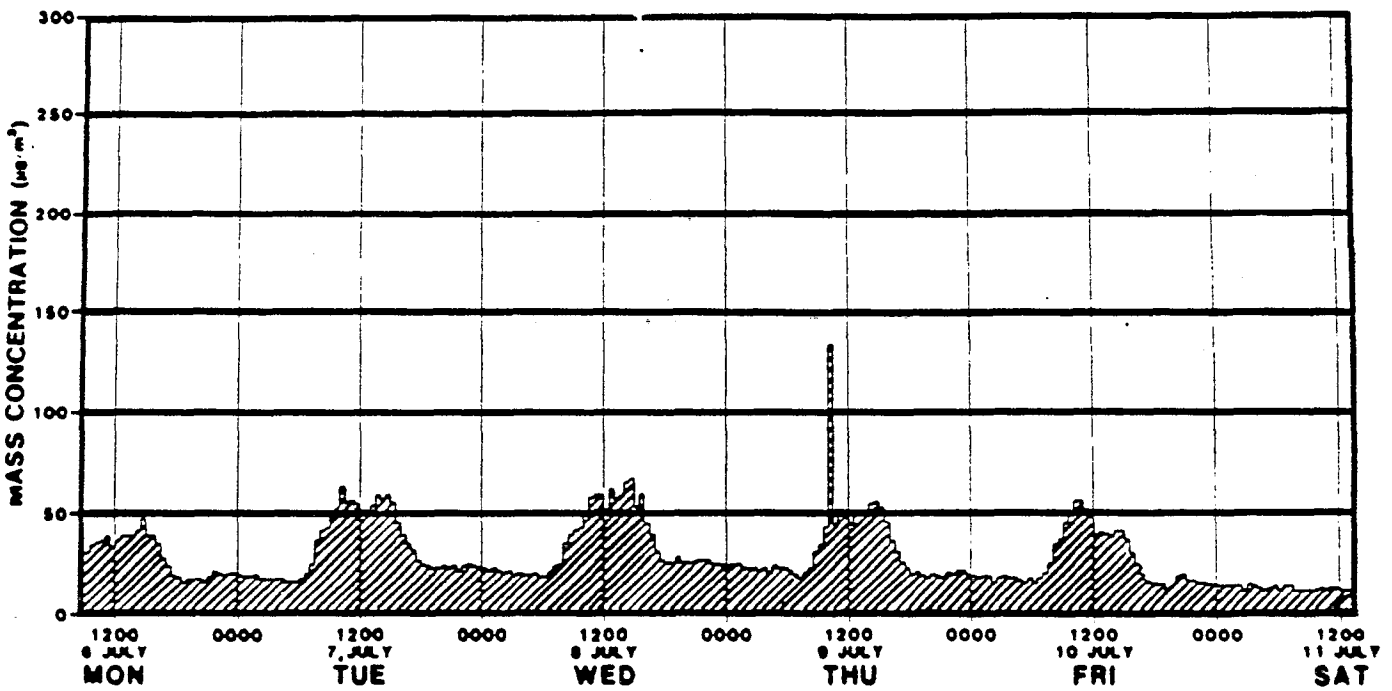

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FIGURE 7. Air exchange rates in commercial buildings are one determinant of nonsmokers' exposures to ETS in the workplace. Shown are air exchange rates (infiltration plus ventilation) in eight large federal office buildings in 8 US cities in 7 states. Air exchange rates in the tighter buildings failed to meet ASHRAE standards for occupied office space, (20 cfm/occ or 0.84 ach). The mean annual air exchange rate for all eight office buildings is 0.71 ± 0.25 air changes per hour, about one third less than in the sample of homes in fig. 4a. (Grot and Persily, 1986).

(4)

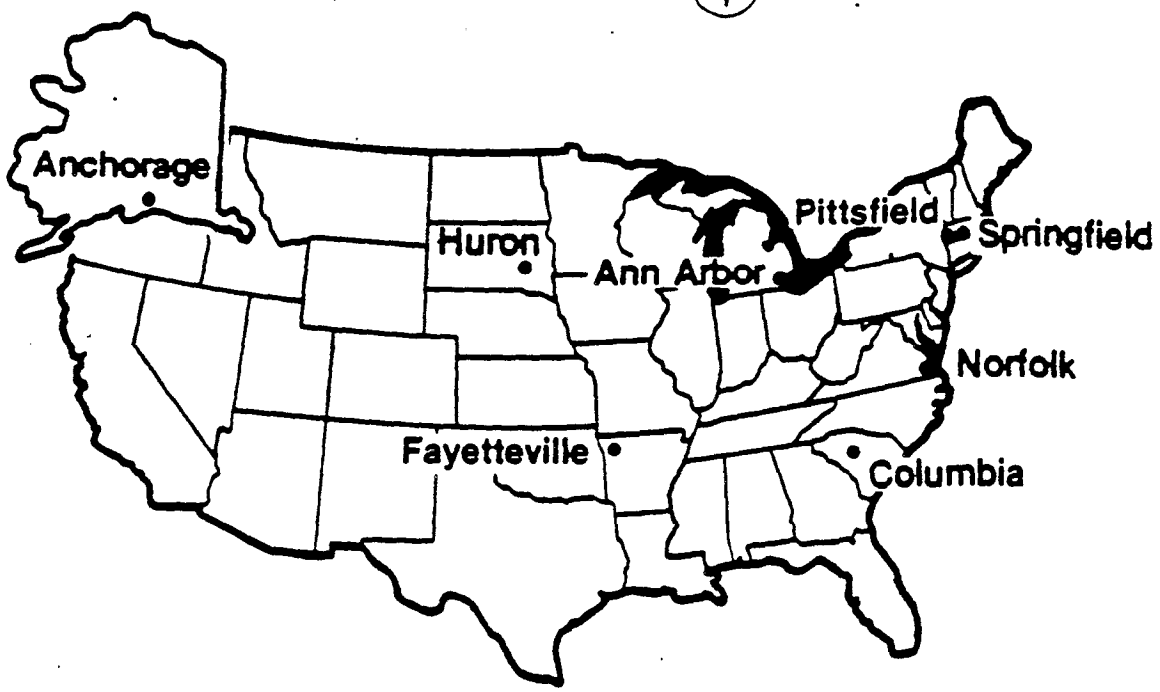

FIG. 1—*Location of the eight federal office buildings.*

BUILDING DIMENSIONS (100 m^2 ≅ 1000 ft^2)

Location	Occupiable Floor Area, m^2	Volume, m^3	Air exchange rate (ach)
Anchorage	45 500	174 000	0.82 ± .35
Ann Arbor	4 900	31 700	1.04 ± .69
Columbia	24 700	159 000	0.85 ± .23
Fayetteville	3 400	21 300	0.37 ± .09
Huron	6 420	27 500	0.32 ± .16
Norfolk	17 300	60 300	0.79 ± .19
Pittsfield	1 730	8 520	0.70 ± .19
Springfield	13 500	57 700	0.79 ± .18

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TABLE 8. (NRC, 1986)

Particulate Levels Measured in Indoor Environments, Including Smoking and Nonsmoking Occupancy

Study	Type of Premise	Occupancy	Volume, m	Ventilation Type Rate	Monitoring Type Time	Concentrations Mean (range) μg/m³	Comments
Brunekreef and Boleij, 1982	4 residences	NS	—	N —	G 2 mo	55 (20-90)	TSP, repeat measures 0.2 m/s
	7 residences	S = 1	—	N —	G 2 mo	125 (60-250)	TSP sensitivity
	14 residences	S = 2	—	N —	G 2 mo	152 (60-340)	TSP sensitivity
	1 residence	S = 3	—	N —	G 2 mo	335 (—)	TSP sensitivity
	Outdoors	—	—	—	G 2 mo	— (41-73)	
Cuddeback et al., 1976	2 taverns	S = 5-40 NS = 5-260 T = 10-300	—	N,M 1-6 ach	G 9 h	446 (233-986)	TSP, ventilation estimated
Elliot and Rowe, 1975	3 arenas	NS	—	—	G 24 h	55 (42-92)	TSP
	3 arenas	S T = 2,000-14,2[illegible]	—	M —	G 0.3 h	350 (148-620)	TSP
First, 1984	1 school	NS	—	M —	P —	20 (—)	TSP
	8 public buildings	S	—	N,M —	P —	260 (40-660)	TSP
Hawthorne et al., 1984	11 residences	NS	150-674	M 0.18-0.96	QCM/5-15 min (over 6 h)	9-40 (—)	RSP, winter/summer—no sources
	8 residences	NS	150-674	M 0.26-1.98	QCM/5-15 min (over 6 h)	12-46	RSP, winter/summer—sources[c]
	2 residences	S	150-674	M 0.27-1.47	QCM/5-15 min (over 6 h)	96-106	RSP, winter/summer—sources[c] + cig.
Leaderer et al., personal communication	3 public buildings	NS	163-1,326	M/0.37-5.6[d]	G/4-21 h	17.8 (9.1-32.2)	TSP, repeat measures, all var.
	7 public buildings	1.7-4.57[b] T = 2-6	168-600	M/0.[illegible]-7.53[d]	G/2-24 h	205.1 (58-452)	Measured (160.0 peak)
Moschandreas et al., 1981	Outdoors	—	—	—	G 24 h	17.0 (—)	RSP, TSP also measured
	2 offices	—	—	—	G 24 h	16.8-20.2 (53 peak)	RSP, TSP also measured
	5 residences	NS T = 2-6	—	N 0.5-1.3 ach	G 24 h	19.4-4.01 (118.9 peak)	RSP, TSP also measured
	5 residences	S [illegible]	—	N 0.5-1.3 ach	G 24 h	36.9-89.9	RSP, TSP also measured
Nitschke et al., 1985	Outdoors	—	—	—	G 168 h	11.3 ± 6.0 (1-28)	RSP
	19 residences	NS	315-1,021	N —	G 168 h	26.0 ± 22.6 (6-88)	RSP, repeat measures, source mix[e]
	11 residences	S	290-800	N —	G 168 h	59.2 ± 38.8 (10-144)	RSP, repeat measures, source mix[e]
Parker et al., 1984	1 residence	NS T = 3	—	N 0.2-1.9 ach	0 24 h	< 10 (—)	TSP
	2 residences	S = 1-2 T = 3-4	—	N 0.2-0.[illegible] ach	0 24 h	10-46 (—)	TSP
Repace and Lowrey, 1980, 1982	Outdoors	—	—	—	P 2 min	42.9 (22-63)	RSP, average of 2-min samples
	27 Public buildings	0.13-3.54[f]	—	M —	P 2 min	278 (86-1,140)	RSP, average of 2-min samples
Sexton et al., 1984	Outdoors	—	—	—	G 24 h	17.0 ± 1.6 (6-23)	RSP, repeat samples
	19 homes						
	24 residences	NS[f]	—	N —	G 24 h	25.0 ± 1.0 (13-63)	Used fireplaces
Spengler et al., 1981	Outdoors	—	—	—	G 24 h	21.1 ± 11.9 (—)	RSP, repeat measures
	35 residences	NS	—	N —	G 24 h	24.1 ± 11.6 (—)	RSP, repeat measures
	15 residences	S = 1	—	N —	G 24 h	36.5 ± 14.5 (—)	RSP, repeat measures
	5 residences	S = 2	—	N —	G 24 h	70.4 ± 42.9 (—)	RSP, repeat measures
Spengler et al., 1985	Outdoors	—	—	N —	G 24 h	18 ± 2.1 (—)	RSP, repeat measures
	73 residences	NS	—	—	G 24 h	28 ± 1.1 (—)	RSP, repeat measures
	28 residences	S	—	—	G 24 h	74 ± 6.6 (—)	RSP, repeat measures
Sterling and Sterling, 1983	1 office	S restr	—	—	G(?) —	25.5 (15-36)	TSP
	22 offices	S	—	—	G(?) —	31.7 (—)	TSP

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TABLE 8. contd.(NRC, 1986)

Study	Type of Premise	Occupancy	Volume, m^3	Ventilation Type Rate	Monitoring Type Time	Concentrations Mean (range), $\mu g\ m^{-3}$	Comments
U S Department of Transportation, 1971	8 domestic planes	S T = 2[a]-110	—	M —	G 1-1 4, 2-1 2 h	Not given (—)	TSP
	20 military planes	S T = 165-219	—	M —	G 6-[b] h	<10-120 (—)	TSP
Weber and Fischer, 1980	44 offices	S	—	N,M —	P 2 min (30 ea)	133 ± 130 (962 peak)	RSP, minus background level

[a]Active smokers per 100 m^3.
[b]Grams of tobacco consumed.
[c]Some smoking was reported during 9 of the 280 samples
[d]Measured during 24-h periods by the perfluorocarbon tracer technique.
[e]Some residences had combinations of sources (kerosene heaters, wood stoves, etc.) and no cigarettes.
[f]Active smokers density per 100 m^3.

ABBREVIATIONS

ach = Air changes per hour
G = Gravimetric
M = Mechanical ventilation
N = Natural ventilation
NS = No smokers
O = Optical monitor
P = Piezoelectric balance
QCMI = Quality Crystal Microbalance Cosade Impactor
RSP = Respirable suspended particles
S = Smokers
T = Total occupants
TSP = Total suspended particles
restr. = building with smoking restrictions

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FIGURE 8.(IARC, 1987)

ndoor a r pollution from ETS aerosol Generally indoor levels of RSP in buildings where tobacco is smoked will greatly exceed those found in buildings where tobacco is not smoked In buildings A-T where tobacco was smoked, the USA National Ambient Air Quality Standards (NAAQS) levels for total suspended particulates (TSP) were exceeded

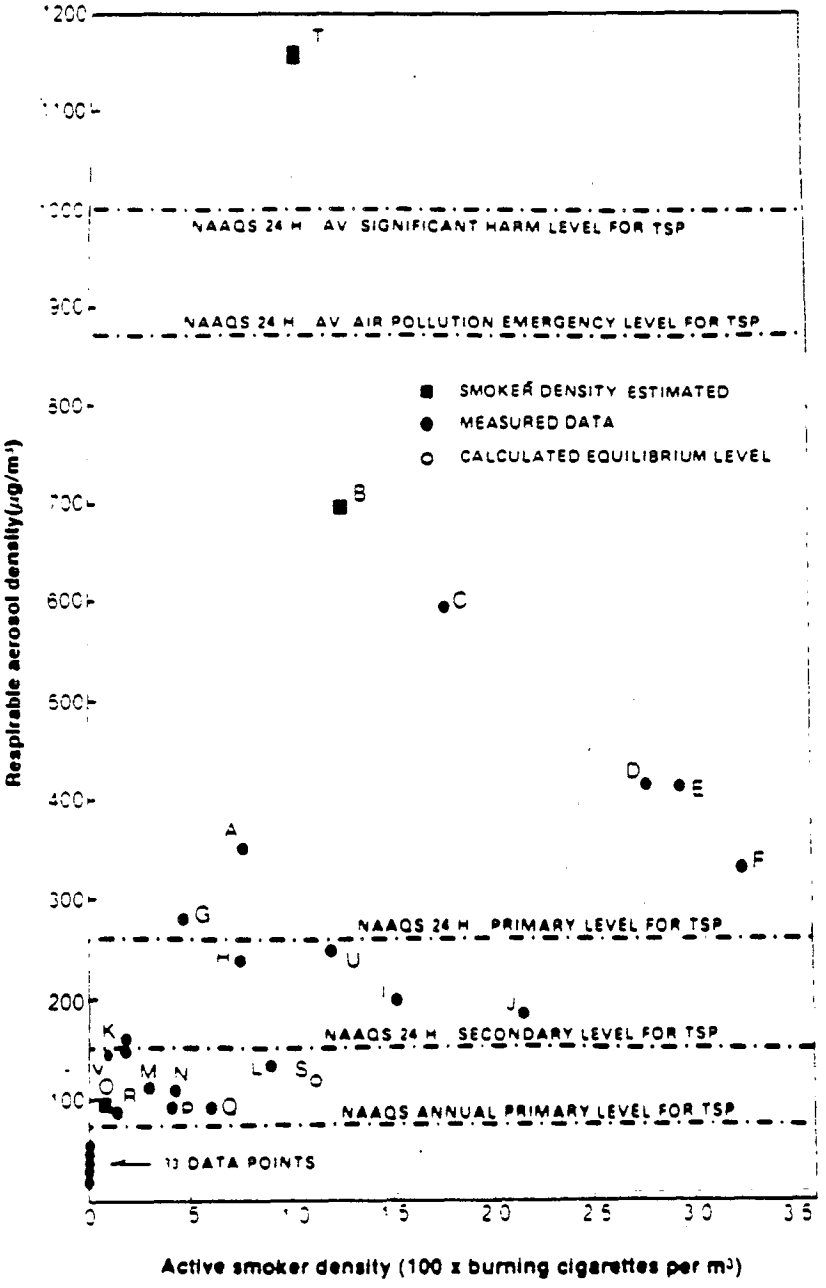

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TABLE 9. Number of persons 17 years and over, by cigarette smoking status, race, sex, and age: United States, 1980. (Unpublished data, National Center for Health Statistics, R. Wilson, personal communication)

Race, sex, and age	Total population	All smokers	Current smokers		Former smokers		Never smoked	All occasional smokers
			Regular and occasional smokers	Regular smokers only	Regular and occasional smokers	Regular smokers only		
	In thousands							
All races[2]								
Both sexes 17 yrs. and over	160,798	86611	52442	51770	33130	30751	74086	3478
17-24 years	30,176	13286	10067	9827	3009	2672	13890	652
25-44 years	61,042	35258	22916	22656	11935	11167	25754	1269
45-64 years	43,556	27170	15236	15236	11477	10571	16330	1141
65 years and over	24024	10896	4121	4050	6667	6317	13112	421
Male 17 years and over	75970	49048	27751	27495	20672	19297	26906	1994
17-24 years	15699	6640	5018	4866	1504	1393	9059	402
25-44 years	29549	19696	12591	12505	6886	6393	9837	692
45-64 years	20839	16238	8702	8557	7612	7113	4593	645
65 years and over	9881	6473	1760	1718	4670	4488	3413	254
Female 17 years and over	84828	37563	24690	24325	12458	11454	47180	1484
17-24 years	14477	6646	5051	4961	1504	1379	7832	249
25-44 years	31493	15562	10345	10152	5099	4774	15917	577
45-64 years	22725	10933	6733	6680	3862	3423	11737	499
65 years and over	14183	4423	2361	2333	1993	1858	9694	163
White								
17 years and over	139086	76041	45090	44515	30197	27976	62910	3045
17-24 years	27095	11525	8588	8400	2758	2463	15569	566
25-44 years	51887	30336	19923	19211	10728	9955	21471	1063
45-64 years	38470	24160	13383	13284	10489	9622	14268	1034
65 years and over	21635	10019	3676	3619	6231	5935	11600	382
Male	65941	43124	23654	23435	19025	17732	22802	1735
Female	73088	32917	21436	21079	11172	10244	40108	1310
Black								
17 years and over	16767	8314	5903	5831	2209	2075	8439	354
17-24 years	4094	1451	1264	1225	158	142	2643	* 56
25-44 years	6584	3656	2657	2624	874	829	2928	159
45-64 years	4891	2471	1636	1636	785	711	1586	* 103
65 years and over	2018	736	346	346	391	369	1282	* 20
Male	7465	4512	3136	3078	1258	1192	2953	184
Female	9302	3802	2767	2753	950	883	5486	166

out of 125,000 — from n 2015

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Source: https://www.industrydocuments.ucsf.edu/docs/jtdl0000

TABLE 10.(Repace and Lowrey, 1985)

Time spent in various microenvironments by persons in 44 U.S. cities, expressed in average hours per day. (Ott, in press; NRC, 1981; Szalai, 1972).

Microenvironment	Employed Men, All Days	Employed Women, All Days	Married Housewives, All Days
Inside one's home	13.4	15.4	20.5
Just outside one's home	0.2	0.0	0.1
At one's workplace	6.7	5.2	–
In transit	1.6	1.3	1.0
In other people's homes	0.5	0.7	0.8
In places of business	0.7	0.9	1.2
In restaurants and bars	0.4	0.2	0.1
In all other locations	0.5	0.3	0.3
Total	24.0	24.0	24.0

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Source: https://www.industrydocuments.ucsf.edu/docs/jtdl0000

reported

Table 11 COTININE IN NONSMOKERS FROM DOMESTIC AND NONDOMESTIC EXPOSURES.(NRC, 1986) 91% of the ETS exposure of the nonsmoking husbands of smoking wives came from non-domestic sources compared to 71% of the exposure of the nonsmoking husbands of smoking wives. The most probable non-domestic source of exposure is the workplace.

TABLE 12-2 Urinary Cotinine Concentration and Number of Reported Hours of Exposure to Other People's Tobacco Smoke Within the Past 7 Days in Nonsmoking Married Men According to Smoking Habits of Their Wives

Smoking Category of Wife	No. of Men	Urinary Cotinine Concentration, ng/ml		Exposure to Other People's Smoke in Preceding Week, h			
				Total		Outside Home	
		Mean (SE)	Median	Mean (SE)	Median	Mean (SE)	Median
Nonsmoker	101	8.5(1.3)[a]	5.0	11.0(1.2)[b]	6.5	10.0(1.2)[c]	6.0
Smoker	20	25.2(14.8)	9.0	23.2(4.1)	21.1	16.4(3.3)	10.7

NOTE: Differences (nonsmoking wife versus smoking wife): [a] $p < 0.05$; [b] $p < 0.001$; [c] $p < 0.06$ (Wilcoxin rank sum test).

SOURCE: Wald and Ritchie (1984).

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TABLE 12. (SG, 1986).

—Cotinine measures in nonsmokers with environmental smoke exposure and comparisons with active smoking

Study	Number of subjects	Smoking status	Exposure level	Mean or median concentration and range: Plasma cotinine (ng/mL) Before	Plasma cotinine (ng/mL) After	Urine cotinine (ng/mL) Before	Urine cotinine (ng/mL) After	Saliva cotinine (ng/mL) Before	Saliva cotinine (ng/mL) After
Jarvis et al. (1983)	7	NS	Before, 11:30 a.m. After, public house x 2 hr	1.1	7.3	4.8	12.9	1.5	8.0
Jarvis et al. (1984)			Hospital clinic patients						
	46	NS	No exposure	—	0.8	—	1.5	—	0.7
	27	NS	Little exposure	—	1.8	—	6.5	—	2.2
	20	NS	Some exposure	—	2.5	—	8.6	—	2.8
	7	NS	Lot of exposure	—	1.8	—	9.4	—	2.6
	94	S		—	275	—	1391	—	310
Hoffmann et al. (1984)	10	NS	Experimental chamber						
			2 cigs burned	1.7	2.6 (peak	14	21	1.2	2.3
			3 cigs burned	1.0	3.0 change)	14	38	1.7	2.5
			4 cigs burned	0.9	3.3	14	56	1.0	1.4
Wald and Ritchie (1984)	101	NS	Wife abstinent	—	—	8.5 (median 5.0)			
	20	NS	Wife smoker	—	—	26.2 (median 9.0)			

Study	Number of subjects	Smoking status	Exposure level	Mean or median concentration and range: Plasma cotinine (ng/mL) Before	Plasma cotinine (ng/mL) After	Urine cotinine (ng/mL) Before	Urine cotinine (ng/mL) After	Saliva cotinine (ng/mL) Before	Saliva cotinine (ng/mL) After
Wald et al. (1984)			Med screening clinic patients						
	221	NS	Research colleagues			—	11.2		
	43	NS	0–1.5 hr ETS exposure/wk			—	2.8		
	47	NS	1.5–4.5 hr ETS exposure/wk			—	3.4		
	43	NS	4.5–8.6 hr ETS exposure/wk			—	5.3		
	43	NS	8.6–20 hr ETS exposure/wk			—	14.7		
	45	NS	20–80 hr ETS exposure/wk			—	29.6		
	131	S	Cigarettes			—	1645 (537–3326)		
	59	S	Cigars			—	396 (61–2138)		
	42	S	Pipes			—	1920 (1008–4569)		
Matsukura et al. (1984)	200	NS	No home exposure			—	510[1]		
	272	NS	All home exposure			—	790		
			Home exposure:						
	25	NS	1–9 cig/day			—	310		
	57	NS	10–19 cig/day			—	420		
	99	NS	20–29 cig/day			—	870		
	38	NS	30–39 cig/day			—	1030		
	28	NS	>40 cig/day			—	1580		
	472	NS	All			—	680		
	392	S	All			—	8620		
	76	NS	No workplace exposure			—	220		
	201	NS	Workplace exposure			—	720		

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TABLE 12. cont'd. (SG, 1986).

Study	Number of subjects	Smoking status	Exposure level	Mean or median concentration and range: Plasma cotinine (ng/mL) Before	Plasma cotinine (ng/mL) After	Urine cotinine (ng/mL) Before	Urine cotinine (ng/mL) After	Saliva cotinine (ng/mL) Before	Saliva cotinine (ng/mL) After
Greenberg et al. (1984)	32	NS, infants	S mother			—	351 (41-1885)	—	9 (0-25)
	19		NS mother			—	4 (0-125)	—	0 (0-3)
Jarvis et al. (1986)	269	NS	Children aged 11-16 Neither parent SM					—	0.4 (median 0.2)
	96	NS	SM father						1.3 (1.0)
	76	NS	SM mother					—	2.0 (1.7)
	128	NS	Both parents SM					—	3.4 (2.4)
Luck and Nau (1985)	10	NS, neonates	No exposure	—	—	—	0[1] (0-56)	—	—
	19	NS, neonates	Nursed by S mother; no ETS exposure	—	—	—	100 (10-555)	—	—
	10	NS, infants	S mother, not nursed	—	—	—	327 (117-780)	—	—
	9	NS, infants	S mother, nursed; ETS exposure	—	—	—	550 (225-870)	—	—
				Serum cotinine (ng/mL)					
Pattenhall et al. (1985)	20	NS, children	Smokers in home	—	4.1	—	—	—	—
	18	NS, children	No smokers in home	—	1.0	—	—	—	—

Study	Number of subjects	Smoking status	Exposure level	Mean or median concentration and range: Plasma cotinine (ng/mL) Before	Plasma cotinine (ng/mL) After	Urine cotinine (ng/mL) Before	Urine cotinine (ng/mL) After	Saliva cotinine (ng/mL) Before	Saliva cotinine (ng/mL) After
Creaton et al. (1980)	68	NS aged <6	No smokers in home	—	—	—	—	—	0, 1.7[2]
	41	NS aged <6	1 smoker in home	—	—	—	—	—	3.8, 4.1
	21	NS aged <6	2 or more smokers in home	—	—	—	—	—	5.4, 5.6
	200	NS aged 6-17	No smokers in home	—	—	—	—	—	0, 1.3
	96	NS aged 6-17	1 smoker in home	—	—	—	—	—	1.8, 2.4
	26	NS aged 6-17	2 or more smokers in home	—	—	—	—	—	5.3, 5.6
	316	NS aged >17	No smokers in home	—	—	—	—	—	0, 1.5
	60	NS aged >17	1 smoker in home	—	—	—	—	—	0.6, 2.8
	12	NS aged >17	2 or more smokers in home	—	—	—	—	—	0, 3.7

[1] ng/mg creatinine.
[2] median, mean.

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TABLE 13. Estimated average nonsmokers' exposures to RSP from ETS at home and at work.(Repace and Lowrey, 1985) The concentrations are calculated for model home and workplace microenvironments and are weighted by average respiration rates and time budget-studies for percent of time spent at home and at work by male and female nonsmokers. The typical nonsmoker is estimated to be exposed to from 0 to 14 mg of RSP from ETS per day, with an average exposure of 1.5 mg/day.

Lifestyle: Daily Average Probability of Being Exposed (Rounded Values)		Exposure (mg) Modeled Daily Average	Exposure (mg) Daily Probability-Weighted
At work and at home: %	63 × 62 = 39	2.27	0.89
Neither at work nor at home: %	37 × 38 = 14	0.00	0.00
At home but not at work: %	62 × 37 = 23	0.45	0.10
At work but not at home: %	63 × 38 = 24	1.82	0.44
Total: %	100		1.43

The average nonexclusive probability of a nonsmoker being exposed to ETS at work is estimated as 63%; the probability of not being exposed at work is 37%; the nonexclusive probability of being exposed to ETS at home is estimated as 62%; the probability of not being exposed at home is 38%.

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FIGURES AND TABLES FOR CHAPTER 7

CHAPTER 8

ABSORPTION OF SMOKE CONSTITUENTS BY NONSMOKERS

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INTRODUCTION

Exposure to environmental tobacco smoke (ETS) occurs at the worksite, in public places, and in private homes. ETS is a composite of effluents generated in various ways during the burning of tobacco products. The major source for ETS is sidestream smoke (SS) which is formed during smouldering of cigarettes, cigars and pipes between the taking of puffs. Minor contributions to ETS are made by those pollutants of the mainstream smoke (MS) that are exhaled after inhalation of each puff by the active smoker. The smoke escaping into the air from the burning cone and from the mouthpiece of a tobacco product during and after puff-drawing is another minor contributor, and there is some diffusion of MS gas phase components through the cigarette paper into the environment.

In the laboratory, MS and SS are generated under standardized conditions by machine smoking (1,2). While these conditions enable us to compare the yields of individual smoke constituents from various brands of cigarettes, cigars and pipe tobacco, they do not fully reflect the patterns of smoking by humans (3,4). The consumer's intensity of puff-drawing and inhaling of the smoke is profoundly influenced by the nicotine content of the MS (4,5), and smoking intensity is highest when cigarettes with perforated filter tips are being smoked (6).

The SS release is governed by the velocity of air currents around the burning cone; thus, higher air flow generates higher yields of most SS components. Even though a major reduction of mainstream smoke yields of the sales-weighted average cigarettes has occurred during the last three decades, (U.S. cigarettes declined from 35.5 mg tar in 1954 to 12 mg tar in 1983; 7), the SS emissions of smoke constituents were not significantly reduced (8,9). The data in Table 1 emphasize this with a comparison of the yields of a select group of toxic compounds in the MS and SS of four types of U.S. cigarettes. These cigarettes were

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machine- smoked under identical conditions. Since the consumer of the low- yield filter cigarettes is likely to smoke more intensely, a larger portion of the tobacco column is burned during smoking of this type of cigarette than is burned during smoking of nonfilter cigarettes. Therefore, a somewhat lower yield of SS is expected from the low-yield cigarette smoked bv the consumer than is obtained by its standardized machine smoking.

The exposure of nonsmokers to the effluents of burning tobacco products usually occurs after considerable dilution of these air pollutants. This is well substantiated by analyses of the air in enclosed spaces polluted by tobacco smoke (10,11).

A. Biological Markers in Physiological Fluids

The exposure of nonsmokers to ETS can be assessed with the help of questionnaires, by estimating the dose from the chemical analysis of smoke-polluted air, by personal monitoring of ETS components and/or by measuring the uptake of individual smoke components in physiological fluids of individuals during or after exposure. The last and most promising method will be discussed in this chapter.

The degree of exposure to ETS depends on several factors, including length of time spent in a smoke-polluted area, the number of smokers within this area, the size and nature of the space, the degree of ventilation and the respiratory rate of the exposed individual. Thus, optimal assessment of ETS exposure is achieved by analysis of physiological fluids of exposed individuals rather than by analysis of the respiratory environment. New biochemical methods enable us to quantify exposure to ETS by determining the uptake of certain smoke contstituents (or their metabolites) in biological fluids. An ordinary requirement for such biochemical measurements is the availability of highly sensitive methods. These should be specific enough for quantitating exposure without interference by other factors.

1. Nicotine and Cotinine.

Disregarding accidental or occupational exposure to tobacco (12,13), or the use of nicotine-containing chewing gum or nicotine aerosol rods as aids for smoking cessation (14), the presence of nicotine and of its major metabolites in physiological fluids is entirely due to the exposure to tobacco, tobacco smoke, or ETS. Nicotine and its major metabolite, cotinine, in saliva, blood or urine of active smokers and of passively exposed nonsmokers are primarily determined by gas chromatography (GC) with a nitrogen-sensitive detector, and by radioimmunoassay (RIA) (15-17). An HPLC method which has been developed for quantitation of cotinine in plasma or saliva of

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smokers (18) has not been applied to urine analysis even though the analysis of this biological fluid appears to have the greatest potential for evaluation of nicotine uptake by nonsmokers. A recently published, highly sensitive method for determining nicotine in plasma by HPLC with dual electrochemical detection (2 ng/ml) has not as yet been applied to physiological samples of involuntary smokers (19). Another emerging analytical method for the determination of nicotine or cotinine is the enzyme-linked immunosorbent assay (ELISA; 20). While the latter two methods appear to be suitable for assays in smokers, they have not yet attained the sensitivity necessary for evaluation of uptake of ETS obtained in current GC or RIA analyses.

Trans-3'-hydroxycotinine has been found to be the most abundant nicotine metabolite in the urine of active smokers (21), however, it is difficult to quantitate this compound. Since the compound is not readily soluble it has to be transformed into a heptafluoro derivative prior to GC detection (22). The levels of 3'-hydroxycotinine in plasma have been found to be much lower than those of cotinine in the same smokers although the renal excretion of 3'-hydroxycotinine has been reported to be greater (23). Despite its abundance in urine of smokers, this compound has not yet been applied to the analysis of ETS uptake by nonsmokers.

The GC and RIA methods are most widely used for assaying nicotine and cotinine in active as well as in passive smokers, primarily because of their specificity and sensitivity, and because the needed instrumentation is available in most modern laboratories. Chromatographic methods, especially those using GC with nitrogen-phosphorus detectors (detection limit 0.1 ng/ ml fluid; 16), or a mass-spectral detection system, offer greatest specificity and high sensitivity; however, they require expensive instrumentation and technical expertise and they are rather labor intensive. Since the air as well as glassware in laboratories may contain traces of nicotine, the chromatographic methods require the utmost precautions to avoid contamination of samples.

The RIA techniques are operationally simpler, less expensive and require smaller samples (detection limit 0.35 ng/sample; 17). More than 50 nicotine metabolites and structurally-related molecules have been tested as inhibitors of nicotine and cotinine antigen-antibody reactions; none of them interfer with the RIA (24). Nevertheless, the potential for cross-reactivity with unknown endogenous components exists. The fact that, upon analysis, thousands of samples obtained from nonsmokers in the US and UK have been found to be negative, indicates that diets and drugs commonly used in these two countries do not pose problems of interference. There is good correlation between results obtained by GC and RIA analysis for plasma cotinine concentrations (r=0.99; 25).

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An interlaboratory comparison of data from 11 laboratories in 6 countries has demonstrated that GC and RIA techniques can reliably quantitate nicotine and cotinine in urine and plasma samples. An excellent correlation of laboratory methods was observed in plasma samples and in urine samples to which cotinine had been added as a tracer. However, in urine samples without tracer, the RIA values for cotinine were found to be slightly higher than those observed by GC. This could be due to a cross reaction of the antibody with another compound present in urine, or the discrepancy could arise from a loss of urinary cotinine during GC extraction. The former explanation is more likely to apply here. All methods have led to perfect distinction between nonsmokers and active smokers (26). Table 2 presents data from model studies on the uptake of ETS by nonsmokers under acute exposure conditions (27-30). The main purpose of these assays was to develop the methodology for field studies and to compare the uptake of nicotine from environments with various degrees of pollution and different types of pollutants under controlled conditions. It has been shown that the equilibrium of nicotine between vapor phase and particulate phase of ETS depends greatly on the concentration and pH of the emitted smokestream (31) and, thus, influences the uptake of nicotine by inhalation.

After repeated exposure to ETS under controlled conditions, such as twice daily 80-minute exposure on 3 consecutive days to the diluted pollutants of 4 concurrently smoked cigarettes (32), the measurements in 4 nonsmokers have shown that except for nicotine in the saliva, the physiological fluids do not reflect maximal concentrations of nicotine and cotinine until at least 24 hours later. This observation has led to comparisons of the elimination of cotinine in smokers and nonsmokers exposed to ETS (33). In the first study, the half-life ($t_1/_2$) of cotinine elimination from plasma of smokers was 18.5 hours; in the case of passive smokers, it was 49.7 hours. The corresponding disappearance ($t_1/_2$)of cotinine from the urine took 21.9 hours and 32.7 hours, respectively. In a second assay, five cigarette smokers were asked to abstain from tobacco use for 5 days and were then given nicotine gum for three days. After another 8 days of abstinence from nicotine, the volunteers were exposed to sidestream smoke. At this point, the cotinine elimination ($t_1/_2$) from urine (ng/ml) by smokers took 15.4 hours, by nicotine gum users 18.2 hours, by 8-day exsmokers 27.5 hours, and by the never-smokers 25.6 hours (33). These findings suggest that the residence times of nicotine, cotinine and other tobacco alkaloids, are likely related to the route of nicotine uptake as well as to possible differences in metabolism between smokers and nonsmokers. The longer elimination time for cotinine in nonsmokers has been confirmed by other study groups (35-37), however, the observation has also been challenged (38,39). A longer residence time of nicotine metabolites in nonsmokers could conceivably increase the possibility of endogenous formation of carcinogenic N-nitrosamines (40).

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Most importantly, differences in the elimination times of cotinine from urine preclude a direct extrapolation to "cigarette equivalents of smoke uptake" by comparing the levels of cotinine excreted by active and passive smokers. This has been discussed by some investigators (10).

Table 3 includes comparisons of nicotine and cotinine in physiological fluids of nonsmokers with or without ETS exposure, and of active cigarette smokers in England (41). Data on the uptake of nicotine by involuntary smokers from additional studies are summarized in Table 4 (29,42-54). Most of these studies demonstrate that nicotine and cotinine levels in physiological fluids of involuntary smokers generally amount to 1 percent and reach maximally a few percent of the amounts determined in active cigarette smokers. Data by Matsukura et al. from Japan on the other hand, show exceptionally high levels of cotinine in the urine of passive smokers. This may be due to several factors including differences in the design of studies (26). Aside from differences in methodology one cannot rule out that differences in the uptake and metabolism of nicotine which have been observed in various population groups, and diet may be partially responsible for the exceptional data reported in the Japanese study (47). In fact, a recent finding indicates that the urinary excretion rates of Japanese smokers were significantly different from those determined in adult cigarette smokers in Europe and North America (55). This requires further thorough investigation.

Survey data on exposure at home, in the workplace and on social occasions were collected from 319 employed probands and were correlated with levels of cotinine in a random urine sample. Mean urine/cotinine/creatinine levels were higher for women than for men possibly due to differences in creatinine excretion between the sexes. It is also noteworthy that 94% of the women were employed indoors. Higher levels of urinary cotinine were noted in both men and women who lived with a smoker than in those subjects who did not report living with a smoker (13.3 ± 2.4 vs 5.1 ± 0.4 in men and 13.9 ± 1.9 vs. 5.6 ± 0.6 in women). Differences in the prevalence of exposure at home existed between sexes (males 13.5% vs. females 29.2%). Levels of cotinine found across different exposures indicate that home exposure has a more pronounced effect on urine cotinine than does workplace exposure (Table 5; N.J. Haley et al., unpublished data).

The nicotine uptake by infants due to ETS exposure, caused by smoking mothers or caretakers, appears to be higher than that observed in adult passive smokers. The amount of cotinine excreted in the urine of the infants was correlated with the number of cigarettes smoked by the mother, or caretaker, during the 24 hours preceding the measurement (33). Since all of the

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infants were on similar diets (they were not breastfed), the influence of nutrition may not play as great a role in the case of these infants but differences in the rates of uptake and metabolism of nicotine and/or the urinary excretion rates of cotinine were certainly established. The finding of relatively high uptake of ETS, as determined by nicotine/cotinine concentrations in the urine of infants, is in line with the observation that infants of smokers have higher rates of respiratory infections than infants in nonsmokers' homes (56).

Analytical data on nicotine and cotinine in physiological fluids of nonsmokers can be misleading in a few cases. These pertain to the very light smokers and those nonsmokers who either chew tobacco or use oral snuff. It is possible, though rare, that the very light smoker shows nicotine/cotinine levels approaching those of passive smokers with extremely high ETS exposure. When used in combination with cotinine measurements, COHb analyses can help to differentiate between the two groups. In regular consumers of snuff or chewing tobacco, cotinine levels are comparable to those found in cigarette smokers while thiocyanate levels and COHb values remain low (57).

The determination of nicotine and cotinine in hair has been tried in an attempt to differentiate between active and passive smokers (58). This determination revealed higher nicotine concentrations in the hair of smokers than in the hair of ETS-exposed nonsmokers and documented the absence of cotinine, the major metabolite of nicotine, within the hairshaft of nonsmokers. Hair sampling for determining ETS-exposure of nonsmokers deserves as yet more thorough investigation.

In summary, in the hands of experienced biochemists, the determination of nicotine and, especially, of cotinine in saliva, serum and/or urine in involuntary smokers represents a reliable, specific method for assaying the level of uptake of ETS by nonsmokers. The choice of biological fluid for the quantitation of cotinine depends upon the question asked. For the evaluation of changes in smoking behavior, serum or urine are preferred while saliva is sufficient to determine whether or not a proband is a smoker (59). For studies of ETS exposure, it is often impractical to collect serum by venipuncture, and since nicotine concentration in saliva can be extremely high immediately following ETS exposure, several hours must pass before the concentration of cotinine in saliva is stabilized (30). Also, when large numbers of probands are to be evaluated, it is preferable to avoid invasive procedures which might discourage participation and possibly bias the results.

Measurements of cotinine in urine and saliva have been successfully used to quantitate ETS exposure in large

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populations. Cotinine excretion in urine is independent of pH, while nicotine excretion is greatly influenced by it. At values above pH 6.0, resorption of nicotine from the urine occurs especially during longer residence time in the bladder. Cotinine is not subject to resorption and, as far as it has been investigated, 3'-hydroxycotinine, a second major nicotine metabolite, is also not affected (60).

Quantitation of cotinine in spot urine samples can have methodological problems relative to the volume of urine excreted in any given time period as well as dilution effects. The ideal standard for evaluation of cotinine excretion in urine would be the analysis of a 24-hour urine sample. Since this is impractical in epidemiological studies, random urine samples are usually collected at the time a questionnaire is administered. In this case, the ratio of cotinine to creatinine in a given sample is often used to allow for differences in urine dilution. Urinary creatinine excretion is usually constant from day to day for a given individual, but it does vary among individuals. As a reflection of muscle mass it is generally excreted at about 1 g per day (men, 1.1 to 3.2 g/day; women, 0.9 to 2.5 g/day). In older persons, the excretion of creatinine may decrease to 0.5 g/day. Low levels of creatinine may also be found in dehydrated infants; this necessitates caution in the expression of ng cotinine/mg creatinine in a random sample (35). A recent study with pre-school children has shown that cotinine/creatinine ratios in passively exposed children 'track' over several weeks and reflect questionnaire data on exposure (61).

2. Carbon Monoxide. Carbon monoxide (CO) is formed during the combustion of organic matter including the burning of a tobacco product. It is also produced in vivo during metabolic processes. Endogenous CO results primarily from the breakdown of heme-containing proteins such as hemoglobin. In nonsmokers who are not exposed to industrial pyrolysis products or vehicle emissions, the baseline levels of CO, present in the bloodstream as carboxyhemoglobin (COHb), are generally below 1.5% of the total hemoglobin.

Persons exposed to heavy vehicle emissions can have COHb levels up to about 2.5%. In cigarette smokers, COHb levels were found to average 5.7% in a study of 450 smokers (62) with little difference being noted between smokers of high- or low-yield products. This value is similar to that of 4.7% found in middle aged men in a study by Wald et al. (63).

Carboxyhemoglobin levels are not good indicators of ETS uptake, due to the fact that CO exposure is not limited to tobacco smoke; in addition, the measurement of COHb is relatively insensitive. A study in England did not find significant differences in COHb levels in subjects reporting no exposure, some exposure, or a lot of exposure (64). This was confirmed by

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others (65) and also by a controlled chamber assay (61). One study in which significant elevations of COHb were found used controlled exposure to tobacco smoke at a level of 25 ppm CO for 8 hours. This intense exposure resulted in an average increase of COHb levels by 2.5% (85). However, such results are not applicable to free-living situations in field studies (67).

3. Thiocyanate. Smoke is detoxified in the liver to thiocyanate (SCN-). Measurement of SCN- has been used to differentiate smokers from nonsmokers or, as mentioned earlier, in combination with nicotine-cotinine assays to distinguish smokers from chewers of tobacco. Thiocyanate can also be derived from the diet, cruciferous vegetables being an excellent source (68). The specificity of SCN as a marker of tobacco smoke inhalation is poor and it is generally difficult to distinouish light smokers from nonsmokers. This lack of specificity makes SCN- unsuitable for the evaluation of ETS uptake by nonsmoking subjects.

4. Hydroxyproline. Japanese investigators have studied the excretion of hydroxyproline in persons exposed to ETS as well as in active smokers and in persons exposed to high levels of air pollutants (69). The rationale for these studies is that the inhalation of nitrogen dioxide causes degradation of lung collagen and elastin which results in urinary excretion of hydroxyproline. The investigations of the Japanese group suggested an elevated excretion of hydroxyproline by children of smoking parents as well as by wives of smoking husbands, active smokers, and individuals exposed to vehicle emissions. Since NOx levels in ETS are relatively low by comparison to mainstream smoke or vehicle emissions (56,70,71), such increased elimination of hydroxyproline in passively exposed persons seemed surprising. In fact, another group of investigators has been unable to confirm this finding (72). Additional investigations, under controlled exposure conditions and field studies are needed before this compound can be properly evaluated as a marker for ETS uptake.

5. N-Nitroso-Amino Acids. The occurrence of endogenous nitrosation reactions in cigarette smokers has been demonstrated in several studies. This phenomenon entails the risk of endogenous formation of carcinogenic N-nitrosamines. Endogenous formation of N-nitrosamines has been proven by urinary excretion of the noncarcinogenic N-nitrosoproline (NPRO), N-nitrosothioproline (NTPRO), and N-nitrosomethylthioproline (NMTPRO). Whereas the average excretion of NPRO in nonsmokers amounted to 2.0±1.5 ug/24 hrs, cigarette smokers excreted an average of 7.0±4.0 ug/24 hrs (73-77). In the case of NTPRO, the average urinary excretion by nonsmokers (ug/24 hrs) was 5.9, that by cigarette smokers 8.7 and that of NMTPRO was 5.6 and 8.5, respectively (75). Only two studies have explored the possibility that endogenous formation of N-nitrosamino acids may

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also be increased in involuntary smokers (77,78).

The data for NPRO in the urine of a limited number of involuntary smokers were not different from NPRO data for nonsmokers without ETS-exposure. A carefully designed study with a larger number of passive smokers may prove that the average value for NPRO or, more likely, for NTPRO is higher in ETS-exposed nonsmokers than in those without ETS-exposure. However, it is unlikely that the determination of N-nitrosamino acids in urine would ever lead to an assay for personal dosimetry of ETS-exposure.

6. Aromatic Amines. The sidestream smoke of cigarettes contains significantly larger quantities of aromatic amines than the mainstream smoke. For example, the MS of a nonfilter cigarette contains 0.36 ug aniline and 0.16 ug of 2-toluidine, whereas the SS of the same cigarette releases 10.8 ug of aniline and 4.1±3.2 ug of 2-toluidine (79). The urine of cigarette smokers contains somewhat higher amounts of aromatic amines than the urine of nonsmokers. The 24-hour urine void of cigarette smokers contains 3.1±2.6 ug aniline and 6.3±3.7 ug 2-toluidine, while the urine of nonsmokers contains 2.8±2.5 ug aniline and 4.1±3.2 ug 2-toluidine (80). The levels of metabolites of these aromatic amines are expected to be markedly higher in the urine of smokers than of nonsmokers. Confirmation of the significance of this difference would encourage the development of analytical dosimetry for evaluation of the impact of ETS-exposure on urinary excretion of the metabolites of aromatic amines.

7. Thioethers in Urine. Cigarette smokers excrete higher amounts of thioethers than do nonsmokers (81). A study of 26 cigarette smokers showed mean urinary thioether values of 4.3±0.4 mmol/mol creatinine compared to an equivalent mean value for 10 nonsmokers of 2.8±0.2 mmol/mol creatinine (82).

In another study nonsmokers were placed on a controlled diet and were subjected to 8-hr ETS-exposure at two levels of concentration. Prior to ETS exposure 10 nonsmokers excreted 40.0±15.4 umol thioethers/24 hrs. The levels rose to 53.9±22.8 umol after exposure to ETS dose 1 (10 ppm CO). At a higher dose level (20-22 ppm CO), pre-exposure values were 69.3±36.3 and post- exposure levels 90.7±44.8. The 10 cigarette smokers who smoked 20 cigarettes each during 8 hrs in order to provide the ETS pollution in the chamber showed an increase of thioether excretion from 89.1±24.8 to 136.1±38.9 umol/24 hrs (67). In other words, the urinary thioether excretion of the passive smokers in this study increased up to 45% and, in the case of the active smokers with the same ETS exposure it increased about 50-65%. These findings require confirmation but they appear to indicate that the thioether analysis of the urine will most likely not be suitable for the determination of the ETS uptake by involuntary smokers.

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B. Genotoxicity of Physiological Fluids

Several studies have explored the possibility that physiological fluids of cigarette smokers contain significantly higher amounts of genotoxic agents than those of nonsmokers (81). The most extensive data base in this field has shown significantly higher mutagenicity in the Salmonella thyphimurium assay of urine of cigarette smokers compared to those of nonsmokers. Since the original study by Yamasaki and Ames in 1977 (83) at least 20 investigations have shown that the urine of cigarette smokers is significantly more mutagenic than the urine of nonsmokers w#o are not ex#osed to genotoxic agents in occupational environments. But it has also been shown that the mutagenicity of the urine of smokers can be effected by diet (84). It has further been surmized that exposure of nonsmokers to ETS may lead to increased urinary excretion of mutagens. Of the 6 published studies in which the urine of passive smokers was tested for mutagenicity with the Ames test, 3 showed increased activity and 3 showed no increase or, at the most an insignificant increase in mutagenic activity (81,85-87). It appears likely that the presently widely used methodology by Yamasaki and Ames (83) can be significantly refined (86,88). This may then enable investigators to assay the urine of involuntary smokers for their exposure to genotoxic agents or their precursors due to exposure to ETS.

C. Adduct Formation of Carcinogens in Passive Smokers.

Measurements in physiological fluids of nicotine and its major metabolite, cotinine, have been shown to be objective indicators of the uptake of ETS. It appears also that, upon refinement of the methodology, the assay for mutagenicity of the urine will reflect the uptake of genotoxic ETS constituents by nonsmokers. However, these assays will not reflect an individual's response to specific ETS carcinogens. That information is best obtained by assessing levels of macromolecular adducts with carcinogens or their metabolites. Development of such assays is based on examining the mechanisms of metabolic activation and detoxification of tobacco smoke carcinogens.

1. Benzo(a)pyrene. In the case of active smokers, adducts of at least 4 types of tobacco carcinogens or procarcinogens have been studied. These adducts are formed by reaction of specific metabolites of tobacco smoke constituents with DNA and/or hemoglobin. Benzo(a)pyrene (BaP), a carcinogenic representative of the polynuclear aromatic hydrocarbons in tobacco smoke is known to be metabolized to bay region diol epoxides (e.g. 7,8-dihydroxy-9,10-epoxy-7,8,9,10-tetrahydroBaP). Such diol epoxides can bind to DNA in human tissues and lymphocytes. Antibodies developed against the major BPDE-DNA adduct have been used to

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assess its presence in surgical specimens of lung tissue, in human placenta, and in peripheral blood lymphocytes (89-91).. Evidence for the presence of such adducts in samples from smokers has been ascertained but significant differences between smokers and nonsmokers have not been observed.

2. Aromatic Amines. 4-Aminobiphenyl and 2-naphthylamine are the known tobacco smoke constituents which are most likely to contribute to the increased risk of bladder cancer of cigarette smokers. The mechanisms by which these compounds are metabolically activated and produce DNA adducts in the bladder epithelium have been extensively studied (92). These studies have shown that the corresponding hydroxylamines are key intermediates in DNA and protein modification. The hydroxylamines also react with hemoglobin, in the case of 4-aminobiphenyl, a sulfinic acid amide of the beta-cysteine (93-95). This adduct readily releases 4-aminobiphenyl upon treatment with dilute acid. A method was developed to analyze the released 4-aminobiphenyl by gas chromatography with detection by negative ion chemical ionization mass spectrometry (95). Application of this method to smokers showed that adduct levels were higher than in nonsmokers, and decreased upon smoking cessation. The method may be further refined for assessing the uptake of carcinogenic aromatic amines from ETS by nonsmokers.

3. Ethylene. This volatile unsaturated hydrocarbon is present in both mainstream smoke (200-400 ug/cigarette) and sidestream smoke of cigarettes (96). Cigarette smoke contains also traces of the carcinogenic ethylene oxide (7.0 ug/cigarette; 97,98). Upon absorption, ethylene is metabolized to the reactive ethylene oxide. The latter binds to cellular macromolecules and to hemoglobin. The alkylated valine is cleaved off of the isolated hemoglobin and the derivatized hydroxyethylvaline is analyzed by GC-MS. Cigarette smokers showed significantly higher hydroxyethylvaline levels (389±138 pg/g hemoglobin) than nonsmokers (58±25 pg/g; 99). So far the method has not been applied to estimates of exposure of involuntary smokers to the procarcinogen ethylene.

4. Tobacco-Specific N-Nitrosamines. During tobacco processing and during smoking tobacco alkaloids give rise to tobacco-specific N-nitrosamines (TSNA). The nicotine-derived N-nitrosamines N'-nitrosonornicotine (NNN) and 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK) are powerful carcinogens. They occur in relatively high concentrations in cigarette mainstream smoke (NNN, 0.12-3.7 ug/cigarette; NNK, 0.08-0.77 ug/cigarette) and sidestream smoke (NNN, 0.15-1.7 ug/cigarette; NNK, 0.2-1.4 ug/cigarette; 40). These agents are metabolically activated by alpha-hydroxylation, leading to a highly reactive intermediate which forms DNA adducts and protein adducts (Fig. I). Metabolic activation of NNN and NNK also leads to the formation of hemoglobin adducts. Acid or base hydrolysis

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of these releases a keto alcohol (compound 5; Fig. I; 100). A highly sensitive GC-MS method has been developed to facilitate the detection of a derivative of compound 5. Refinement towards further increased sensitivity of the method should lead to a dosimetry assay allowing determination of the uptake of the carcinogenic TSNA by passive smokers.

FUTURE NEEDS

The absorption of tobacco-specific smoke constituents from ETS has been demonstrated through analyses of nicotine and its major metabolite, cotinine in the body fluids of exposed nonsmokers. Less tobacco-specific markers have also been measured in exposed populations; however, the results were ambiguous in regard to the quantitative uptake of ETS. There is a need to provide information about the uptake and disposition of carcinogenic constituents by individuals exposed to ETS in acute and chronic situations. Analyses to be fully developed and applied to passive smokers will include measurements of adducts of genotoxic smoke constituents covalently bound to DNA or hemoglobin. These techniques have been developed for benzo(a)pyrene, 4-aminobiphenyl, ethylene, and tobacco-specific N- nitrosamines. It is not known whether or not all of these methods can be made sufficiently sensitive to monitor the uptake of tobacco-specific components from ETS.

Nicotine in ETS is predominantly present in the vapor phase of the smoke rather than bound to the aerosol particles. In order to measure the uptake of carcinogens and toxins residing in the particulate phase of ETS, deposition studies must be developed with specific markers. Particulate phase constituents which could be quantitated include tobacco-specific N-nitrosamines, polyphenols, such as the immunoactive compound rutin, or the tobacco-specific solanesol.(101) However, the levels of these compounds are expected to be low so that development of suitable methodology calls for highly sensitive detection methods.

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TABLES AND FIGURES FOR CHAPTER 8

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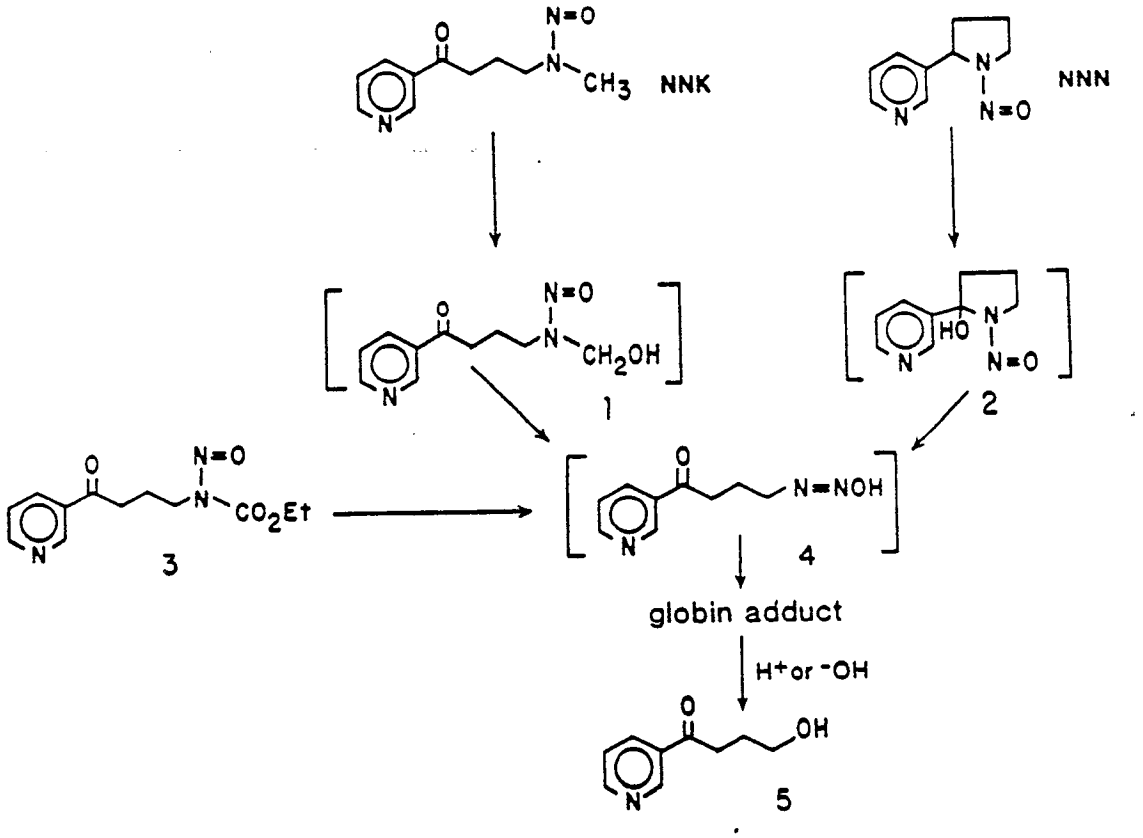

Figure I. Metabolic activation of 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK) and N'-nitrosonornicotine (NNN) to intermediates which bind to DNA and protein.

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Table 1.

Toxic and tumorigenic agents in MS and SS

Smoke Constituent	Smoke stream[a]	Cigarette A (NF)	B (F)	C (F)	D (PF)
Tar (mg)	MS	20.1	15.6	6.8	0.9
	SS	22.6	24.4	20.0	14.1
Nicotine (mg)	MS	2.04	1.50	0.81	0.15
	SS	4.62	4.14	3.54	3.16
CO (mg)	MS	13.2	13.7	9.5	1.8
	SS	28.3	36.6	33.2	26.8
Catechol (µg)	MS	41.9	71.2	26.9	9.1
	SS	58.2	89.9	69.5	117
BaP (ng)	MS	26.2	17.8	12.2	2.2
	SS	67.0	45.7	51.7	44.8
Ammonia (µg)	MS	76.0	19.4	34.0	40.4
	SS	524	893	213	236
NDMA (ng)	MS	31.1	4.3	12.1	4.1
	SS	735	597	611	685
NPYR (mg)	MS	64.5	10.2	32.7	13.2
	SS	117	139	233	234
NNN (ng)	MS	1007	488	273	66.3
	SS	857	307	185	338
NNK (ng)	MS	425	180	56.2	17.3
	SS	1444	752	430	386

[a] Abbreviations: NF, nonfilter cigarette; F, filter cigarette; PF, cigarette with perforated filter tip; BaP, benzo(a)pyrene; NDMA, N-nitrosodimethylamine; NPYR, N-nitrosopyrrolidine; NNN, N'-nitrosonornicotine; NNK, 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone.

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Table 2.

Uptake of nicotine by nonsmokers exposed to ETS under controlled conditions

ETS-Conditions	No. of Passive Smokers	Results	Investigator(s)
Room - 170 m^3 (11 smokers)		Urinary excretion	
(a) 100 cigarettes were smoked during 2 hrs; no ventilation (30 ppm CO)	7	Nicotine: 10±6.8 µg/6 hrs. Cotinine: 35±34.5 µg/6 hrs.	
(b) same conditions as above (a) but with ventilation (5 ppm CO)	7	Nicotine: 18±7 µg/6 hrs. Continine: 19±9.4 µg/6 hrs.	
Room - 66 m^3 (4 cigarette smokers)		Nicotine/Urine (µg/24 hrs)	Cano et al. (28)
(a) Day 1, nonsmoking	2	0 - 0	
" 2, 98 cig's smoked		35 - 44	
" 3, 121 " "		50 - 61	
" 4, 98 " "		62.5 - 70	
" 5, 88 " "		47 - 50	
(b) Day 1, 97 " "	2	23 - 34	
" 2, 96 " "		22.5 - 58	
" 3, 94 " "		47.5 - 69	
" 4, 103 " "		32 - 65	
Room - 43 m^3		Nicotine/Plasma (µg/ml)	Russell and Feyerabend (29)
9 smokers consumed 80 cigarettes + 2 cigars no ventilation (38 ppm CO)	12	Before exposure: 0.73±1.6 After 78 min. exposure: 0.9± 0.29 Nicotine/Urine (ng/ml) 15 min. after exposure: 80.0±58.7	

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Table 2 continued.

ETS-Conditions	No. of Passive Smokers	Results				Investigators
Room - 16 m^3	6	Time during exposure		Nicotine (ng/ml)	Cotinine (ng/ml)	Hoffmann et al., 1984 (30)
4 cigarettes concurrently and continuously smoked for 80 min; 6 air exch./hr. (200 g nicotine/m^3; 20 ppm CO)		0	Saliva	3	1.0	
			Plasma	0.2	0.9	
			Urine	17	14	
		80 min.	Saliva	730	1.4	
			Plasma	0.5	1.3	
			Urine	84	28	
		Time following exposure				
		30 min.	Saliva	148	1.7	
			Plasma	0.4	1.8	
		150 "	Saliva	17	3.1	
			Plasma	0.7	2.9	
			Urine	100	45	
		300 "	Saliva	7	3.5	
			Plasma	0.6	3.2	
			Urine	48	55	

*Nicotine and cotinine were measured in urine as ng/mg creatinine.

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Table 3.

Approximate Relations of Nicotine as a Parameter Between Nonsmokers, Passive Smokers, and Active Smokers[a] (41)

Nicotine/Cotinine	Nonsmokers without ETS Exposure No. = 46		Nonsmokers with ETS Exposure No. = 54		Active Smokers No. = 94
	Mean Value	% of Active Smokers' Value	Mean Value	% of Active Smokers' Value	Mean Value
Nicotine (ng/ml)					
in plasma	1.0	7	0.8	5.5	14.8
in saliva	3.8	0.6	5.5	0.8	673
in urine	3.9	0.2	12.1*	0.7	1,750
Cotinine (ng/ml)					
in plasma	0.8	0.3	2.0*	0.7	275
in saliva	0.7	0.2	2.5**	0.8	310
in urine	1.6	0.1	7.7**	0.6	1,390

[a]Differences between nonsmokers exposed to ETS compared with nonsmokers without exposure: *p<0.01; ** p<0.001.

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Table 4.

Uptake of nicotine by nonsmokers exposed to ETS under daily life conditions 2040225505

Nonsmoker Group	Number of Nonsmokers	Results	Reference
		Nicotine/Urine (ng/ml)	
Hospital personnel	14	12.4±16.9	Russell and Feyerabend (29)
(78 min in smoke-filled room)	13	8.9±9.1	
Hospital personnel and outpatients		Nicotine/Saliva (ng/ml)	
a) no exposure to ETS	26	5.9 7.5	Feyerabend et al. (42)
b) exposed to ETS	30	10.1 21.6	
Flight attendants	6	Nicotine/Serum (ng/ml) pre flight: 1.6±0.8 post flight: 3.2±1.0	Foliart et al. (43)
Office workers a) 11:30 a.m. sample b) 7:45 p.m. sample after 2 hr stay in pub	7	Content/ml: saliva — Nicotine (ng) a)1.90 b)43.63; Cotinine (ng) a)1.50 b)8.04 serum — 0.76 2.49; 1.07 7.33 urine — 10.57 92.63; 4.80 12.94	Jarvis et al. (44)
Hospital staff and outpations		Cotinine/Urine (ng/ml)	Wald et al. (45)
a) no exposure to ETS	22	2.0 (0.0 - 9.3	
b) exposed to ETS	190	6.0 (1.4 -22.0)	

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Table 4 continued ...

Nonsmoker Group	Number of Nonsmokers	Results	Reference
Husbands of		Cotinine/Urine (ng/ml)	Wald and Ritchie, (46)
a) nonsmokers	101	8.5± 1.3	
b) smokers	20	25.2±14.8	
Nonsmokers		Cotinine/Urine (ng/mg creatinine)	Matsukura et al., (47)
a) nonsmokers at home	200	0.5 ±0.09	
b) smokers at home	272	0.79±0.1	
Cigarettes smoked day in home of nonsmokers;		Cotinine/Urine (µg/mg) creatinine)	
1- 9	25	0.31±0.08	
10-19	57	0.42±0.1	
20-29	99	0.87±0.19	
30-39	38	1.03±0.25	
> 40	28	1.56±0.57	
unknown	25	0.56±0.16	

Nonsmoker Group	Number of Nonsmokers	Nicotine/Urine (ng/mg creatinine)	Cotinine/Urine (ng/mg)	Reference
Infants (≤10 months, not breastfed)				Greenberg et al.; (33)
a) not exposed to ETS	18	0 (0-59)	4 (0-125)	
b) exposed to ETS	28	53 (0-370)	351 (41-1,885)	

Nonsmoker Group	Number of Nonsmokers	Results	Reference
School children (11-16 yrs)		Cotinine/Saliva (ng/ml)	Jarvis et al., (48)
a) Neither parent smoked	269	0.44±0.68	
b) Only father smoked	96	1.31±1.21	
c) Only mother smoked	76	1.95±1.71	
d) Both parents smoked	128	3.38±2.45	

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Table 4 continued ...

Nonsmoker Group	Number of Nonsmokers	Results				Reference
Neonates and infants		Nicotine (ng/mg creatinine)		Cotinine		Luck and Nau, (49)
a) No exposure (4-8 days old)	10	(0)	0 - 14	(0)	0- 56	
b) Exposure via breast feeding (3-8 days old)	19	(14)	5 -110	(100)	10-555	
c) Passive smoking (2.5-6 months old)	10	(35)	4.7-218	(327)	117-780	
d) Exposure via breast feeding and passive smoking (1-12 months old)	9	(12)	3.0- 42	(550)	225-870	
Infants (age 3-15 months) exposure in the home		Cotinine/ Serum (ng/ml)				Pattishall et al., (50)
Black infants						
a) no exposure	9	1.0 (1.87±2.38)				Pattishall et al., 1985 (51)
b) passive smoking	15	4.0 (5.27±3.50)				
White infants						
a) no exposure	9	0.0 (0.22±0.44)				
b) passive smoking	5	0.4 (0.90±1.30)				

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Table 4 continued ...

Nonsmoker Group	Number of Nonsmokers	Results						Reference
		Cotinine/Urine (ng/mg creatinine)						
Neonates and infants		No. exp'd	I		No. exp'd	II		Schwartz-Bickenbach et al., (51)
a) Mother smokes, breastfeeds	20	12	(1756)	0 -3520	8	(935)	488-2440	
b) Mother smokes, feeds bottle	16	4	(47)	0 - 160	12	(107)	0- 341	
c) Father smokes	18	10	(0)		8	(0)	0- 308	
d) No exposure in the home	15	9	(0)		6	(0)		

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Table 4 continued ...

Nonsmoker Group	Number of Nonsmokers	Results	Reference
Municipal workers		Cotiine/Urine (ng/mg creatinine)	Sepkovic et al., (52)
I. ETS exposure in the workplace			
a) no exposure	25	4.5±0.6	
b) light expsoure	126	6.6±0.6	
c) moderate exposure	84	7.2±0.8	
d) heavy exposure	32	8.4±1.3	
II. ETS exposure in the home			
a) no exposure	77	6.1±0.8	
b) light exposure	83	6.7±0.6	
c) moderate exposure	71	7.8±1.1	
d) heavy exposure	34	7.6±1.3	
School girls (11-16 yrs) ETS exposure in the home			Jarvis et al., (53)
a) neither parent smokes	104	1.1±0.5	
b) father smokes only	76	2.0±0.6	
c) mother smokes only	40	3.2±0.8	
d) both parents smoke	110	5.0±1.0	

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Table 4 continued ...

Nonsmoker Group	Number of Nonsmokers	Results			Reference
Children and adults	529 males 768 females	Cotinine/Saliva (ng/ml) smokers in family			Coultas et al. (53)
		none	one	≥ two	
a) ≤5 years old		0.0 (0.0-2.5)	3.8 (0.0-6.1)	5.4 (3.2-7.7)	
b) 6-12 years old		0.0 (0.0-2.1)	2.0 (0.0-3.8)	5.2 (1.5-7.0)	
c) 3-17 years old		0.0 (0.0-2.0)	2.9 (0.0-4.9)	4.1 (2.7-7.6)	
d) 18-29 years old		0.0 (0.0-2.6)	0.0 (0.0-5.8)	0.0 (0.0-4.4)	
e) 30-64 years old		0.0 (0.0-2.7)	1.9 (0.0-4.5)	4.4 (1.8-11.0)	
f) ≥ 65 years old		0.0 (0.0-2.6)	3.6 (0.0-6.5)	0.0	

*Numbers in parenthesis median values.

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CHAPTER 9

THE EFFECTS OF PASSIVE SMOKING AND DAY CARE ON RESPIRATORY ILLNESSES IN CHILDREN

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1. INTRODUCTION

Upper respiratory infections are the most common illnesses affecting children under 5 years of age. They are important causes of childhood morbidity and their treatment consumes a substantial portion of health care resources. (8, 14, 17) Infants average 7-8 acute respiratory illnesses per year and older children, 1-5 years of age, average one or two fewer infections. (17)

Acute otitis media (AOM) is the most common complication of upper respiratory diseases in infants and young children. (16, 18, 31, 47) AOM is the largest single cause of morbidity with possible sequelae in children (47) and recurrent episodes are very common in children during the first years of life. (23) AOM account for one-third of pediatric office visits (31) and three-fourths of follow-up visits. (16) Nearly all children have at least one episode of otitis media with effusion (OME) during their first 6 years of life. The majority will recover spontaneously but some will develop chronic OME or chronic otitis media with perforation and discharge. (45) Repeated episodes of OME in early life may lead to developmental or educational delays due to transient or permanent hearing loss and impeded speech. (17, 47)

Bronchiolitis is the most common manifestation of lower respiratory tract infections in infants and small children. The true incidence is unknown but the risk of hospitalization for infants with bronchiolitis is estimated at 10 per 1,000. The mean age for respiratory syncytial virus (RSV) bronchiolitis is 7.8 months and the peak age is 2 months. One half of children hospitalized for the condition are under 3 months of age. (42)

One benefit-cost analysis study (25) concluded that immunization

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against the four most common respiratory viruses would appear to be a reasonable step. However, Denny (9) cautioned that the two commercially available vaccines, S. pneumoniae and type b H. influenzae, are not very effective in children under two years of age where most infections occur. He also noted that while current efforts to produce effective respiratory vaccines are extensive, the problems associated with their development are very great.

2. DAY CARE IN THE U. S.

2.1.GENERAL CHARACTERISTICS

2.1.1. Types

In 1982, 6 million mothers (48.2%) with a child under the age of 5 were in the civilian work force and the upward trend is continuing (28). Their children are cared for in three basic types of day care delivery systems. They are in-home care, family day care, and group day care.

In-home care is provided in the child's home by a parents, relative, or non-relative. In 1982, 30.6% of the children of working mothers received in-home day care. Fathers and others relatives provided most of this type of care. (28, 50) In-home care by a non-relatives is the most expensive form of care and is used by only 5.5% of working mothers. (35)

Family day care (day care homes) is given by a non-relative in a private home other than the child's. (2, 50) Day care homes include more children than any other form of day care. These homes are generally divided into three basic types: informal homes, registered/licensed homes and network homes. Informal homes - which are not regulated, registered, or linked to any sponsoring agency - are the most common. Ninety-four percent (94%) of all children in family day care attend these homes. Registered or licensed day care home are required to meet state/local regulation. The smallest subset of day care homes operate within networks administered by a sponsoring agency that is either for profit or nonprofit. The average number of children per day care home is 3.5. Non-regulated homes average 3.6 children compared to 4.5 in regulated homes. (2, 15)

Day care homes offer a cozier settings with more adult attention and fewer peers. Individual discipline and training for compliance are usually better. They are well suited for the care of infants, toddlers, and slow-to-mature older children. This form of care is also the least expensive. However, most providers have little or no training (17, 35) and there is no assurance that providers place children's needs above the desire to earn a living. (17)

Day care centers, including nurseries, provide care for 12 or more children in nonresidential buildings. (17, 49) They usually offer

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an organized educational program and scheduled activities for children. There is a wide variety of play materials, child-size furnishings, outdoor space and equipment, and a school-like atmosphere. Caregivers are also more likely to have some training in child development. (35) In the typical day care center, children spend a major portion of their time grouped by age in separate rooms and under the supervision of one or more adults. This sector is almost always subject to government regulation and is the smallest of the 3 sectors. (17) However, centers are the fastest growing segment in the day care market. (1, 24, 28)

2.1.2. Usage

The most drastic change in infant care has been the return of parents to work while their children are infants. (26) In a 1982 report, Child Care Arrangements of Working Mothers (28), 6 million women aged 18 to 44 with a child under 5 years of age were in the civilian labor force. Table 1 list the percent distribution of the type of child day care used by the age of the child. Nine percent (9%) of working mothers were able to care for their children while working. Almost one-third (30.5%) arranged for in-home care of their children. However, day care homes were the predominate source of care selected ,i.e., 40.2%. The use of day care centers increases with the age of the child.

Table 2 describes the percent distribution of care-givers by the age of the child. Fathers were the primary care-givers to 14% of the children of working mothers. Among husbands who were the principal caretakers, 71% were employed, 24% unemployed and 5% were not in labor force. (28)

Relatives provided child care to 29% of the children of working mothers. Grandparents were the principal care-givers among relatives. Seventeen percent (17%) of all pre-schoolers and 22.4% of infants received day care from their grandparents. Other relatives cared for 12% of the children. Grandparent or other relative, however, provided care for the children of 40% of unmarried mothers. (28)

Non-relatives provided 27.5% of all day care to children of working mothers. This makes non-relatives the single largest source of care-givers. Data in Table 3 show that care was given in the home of a non-relative to 22% of all children and about 25% of infants and toddlers. (28)

2.1.3. Personnel

In the National Day Care Center Study (33), the average weekly fee in day care homes was $20.85 per child. The fees were $17.80, $22.65, and $26.36 in non-regulated, regulated, and sponsored homes respectively. In both day care homes and many centers, the mean

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wage for care givers was found to be below minimal wage. The discrepancy represents a subsidy for the cost of child care by these care givers. Zigler and Muenchow (50) also reported that 60% of day care workers have income below the poverty level

In 1980, the prevalence of smoking among female child care workers was estimated at 28.9%. Thus child care workers smoked less than females in general. However, their rates are much higher than those for female elementary school teachers (19.8%) and higher than secondary school teachers (24.8%). (44)

2.2.REGULATIONS

2.3.1. Federal

Unlike most other Western nations, the U.S. has no national policy on child care and efforts to develop one have reached a stalemate. (35) Since 1970, there have been several attempts to create federal regulation of at least a portion of the day care market. (17) Minimal conditions for safety were proposed in 1980 but were never enacted. (50) While the 1980 Federal Interagency Day Care Requirements have been withdrawn, they continue to serve as a guideline for minimum standards. (49)

2.3.2. State

Each state regulates its own day care facilities. In most states, the requirements are severely written and laxly enforced. (35) All 50 states have enacted regulations which contain some provisions for health and safety but they vary greatly across the country. (17) Licensing practices also vary from state to state. (24) Forty-four (44) states currently regulate family day care homes. (49) A state license, however, does not guarantee that a day care facility meets minimum state requirements. The under-staffing of state licensing bureaus often makes licensing and monitoring a hasty and sporadic effort. (50) Children cared for in their own home are generally beyond the reach of federal and state policy (17).

In some states, the physical requirements for day care homes rule out most residences as sites for child care, even though most children are reared in these same homes. The regulations were written for day care centers and not homes. Nevertheless, they are applied to homes, which means that many day care homes go unregulated and unlicensed. (35)

This analysis of existing regulations presents a very bleak picture with great variations among the states in standards for day care. The majority of day care facilities are unlicensed. States vary in their policies of periodic inspections, unannounced visits, and procedures for license renewal. (49)

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2.3.RELATIONSHIP TO RESPIRATORY INFECTIONS

Respiratory diseases are the most common illnesses affecting children in day care. (1) Today, infants and pre-school age children acquire infections at earlier ages. The common factor in this changing pattern is the increasing popularity of day-care centers. Today, younger and younger children are spending more and more time outside the home on a daily basis. (24) Day care centers with many children in the same place create favorable conditions for respiratory epidemics, with acute otitis media as a sequelae. (30) However, the total burden of respiratory illnesses appears to be no greater for the day care child. The illnesses simply occur at younger ages. (1)

The association of day care and respiratory tract illnesses began in the 1920's. (17) In the 1970's, Scandinavian researchers (19, 23, 31, 32, 39, 41, 45) found an increased incidence of otitis media and/or recurrent otitis media over prolonged periods among children attending day care. The strongest evidence of an increased rate of otitis media was found among children in centers, with family day care holding an intermediate position between centers and in-home care. Moreover, home-reared children with abnormal finding at first testing were significantly more likely to return to normal at each subsequent testing than were children attending centers.

While their are obvious difficulties in transferring the results from studies on infections in day care facilities conducted in Scandinavian countries, Haskins (17) concluded that because of the high quality of these studies, especially the epidemiologic studies based on entire birth cohorts, the findings deserve careful attention. Thus, Scandinavian studies provide substantial reasons for concern that children in day care centers, and possibly family day care, are at 2-3 time the risk for persistent otitis media as those reared at home

These findings were confirmed in a case-control study by Visscher and colleagues (47) with patients at a large pediatrics group practice in Minneapolis. During a 2-week period in February, 1982, every parent who brought a child into the clinic for any reason was asked to fill out a questionnaire. Cases were defined as presenting with AOM on a study day, regardless of past history. Controls were patients with a diagnosis other than otitis media and who also had no prior history of otitis. A cold in the past week and the symptoms of fever, ear pain, and decreased hearing were prominent in the case group. Attending a day care facility was the second most important risk factor and the risk increased with the number of other children at the facility. Exposure to smokers was not a risk factor. While the study had a number of flaws, 4 bona fide risk factors were identified, i.e., recent upper respiratory tract infection, allergy, day care exposure, and family history of ear infection.

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Fleming and colleagues (14) also found that children in Washington, DC who attended day care were significantly more likely to have an upper respiratory tract infections during a 2-week period prior to the interview. In addition to day care attendance, a second factor, maternal smoking was also associated with an increased risk. The effects of day care attendance and maternal smoking were independent of one another. Age and living in crowded conditions were also risk factors. The researchers estimated that 31% of upper respiratory infections can be attributed to day care attendance.

Most studies of bronchiolitis have been restricted to the hospitalized child. Comprehensive studies of this disease in ambulatory patients or day-care centers are lacking. (42) However, the incidence of bronchiolitis in one day care center in Chapel Hill, NC was compared with the rates in a pediatric practice. The rate was much higher in the day care center for children 6 months of age or younger. However, the proportion of cases requiring medical treatment and hospitalization was less among day care attendees. (10)

Several reviewers (1, 17, 18) of studies on respiratory illness and day care have identified the following problems which limits the generalization of the findings:

1.Control groups were less than satisfactory in that day care children were often observed on a daily basis whereas those in home care were not.

2.Some studies reported respiratory tract symptoms while others used diagnostic categories.

3.There is little commonalty in either the ages of children studied or the manner of reporting illnesses by age category.

4.The reliability of case-controlled and cohort studies depends on the accurate quantification of disease occurrence in members of all study groups. This raises the questions of whether parents of children in day care seek a physician for their children's illnesses more frequently. The suggestion by a day care provider that a child be taken to a physician might have important effects on parents.

Henderson and colleagues (18) warned that the apparent association of day care attendance with otitis media could be accounted for, in part, by these types of bias. Perhaps more importantly, Ingvarsson and colleagues (19) emphasized that most studies have not investigated other factors that probably influence the incidence of respiratory illnesses, i.e., external factors such as type and quality of housing, degree of air humidity and ventilation, and cigarette smoke inhalation and other air

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pollution.

Nonetheless, reviewers have concluded that most studies have shown an increase in mild acute respiratory illnesses among children in day care and this effect occurred primarily in younger children. There is stronger evidence for initial and recurrent otitis media. (17) Large group day care centers and probably smaller day care homes are settings in which the incidence of otitis media is increased for young children. (2, 17, 18, 31) Studies also show reduced rates of both symptoms and acute illnesses with increasing age in all sites and there is very little evidence of an excessive rate of illness in day care children for the more serious respiratory illnesses. (17) Although most studies have found increased rates of acute, minor, respiratory illnesses among children in day care, asymptomatic children in day care do not have higher levels of respiratory tract pathogens or even different pathogens than children reared at home. (1, 17, 40) However, The true incidence of infectious disease in family day care is unknown since most of it is unlicensed. Therefore, conclusions cannot be made on this mode of care. (2)

3. PASSIVE SMOKING

3.1. RELATIONSHIP TO DISEASES IN INFANTS AND CHILDREN

A number of studies have demonstrated a positive association between passive smoking and lower respiratory symptoms (4, 5, 13, 36) and lower respiratory diseases (11, 12, 22, 29, 48) in infants and young children. Maternal smoking, when measured, showed a high correlation with respiratory symptoms and diseases in the above studies whereas, paternal smoking was rarely significant.

Studies on the relationship of passive smoking to the development of bronchiolitis are less clear. Two studies (32, 38) show a positive association with maternal smoking. However, Pedreira and colleagues (29) did not find a relationship.

Otitis media is the only upper respiratory disease reported in the literature as being associated with passive smoking. Six studies (3, 20, 21, 30, 34) showed an increase incidence of otitis media with maternal smoking. However, in four other studies (39, 45, 46, 47) parental smoking was not significant. Pukander and colleagues (30) suggest that these studies may not have found a relationship because of the masking effect of day care attendance and other confounding variables.

Two comprehensive reviews (27, 43) have concluded that bronchitis, pneumonia, other lower respiratory tract illnesses and otitis media occur more frequently during the first year of life in children with mothers who smoke. The National Research Council (27), however, emphasized the need for caution in the interpretation of the results of the studies on the effect of involuntary smoking.

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Independent risk factors, such as age and sex, were not always taken into account. Interpretation of epidemiological studies is hampered by the existence of factors that interact with and modify the response to exposure and by confounding factors that are associated with the same symptom complex. While several statistical approaches were used to control for potentially confounding variables, however, researchers do not agree on the nature of the roles of the variables as confounders and the appropriate ways to introduce these variables into the data analysis. The use of questionnaires to collect information on symptoms are prone to recall bias. Most studies examined only the effects of exposure to parental smoking, excluding exposures outside the immediate family, and found significant association only for the effect of maternal smoking. In future studies, great care must be taken to account for potential confounding variables in the analyses.

Two researchers offered explanation for the observed association with only maternal smoking. Pedreira and colleagues (29) postulated that the mother, more often than the father, remained at home with the child. This also suggest that the duration of exposure rather than the presence of a smoker is the important factor in infant-related respiratory diseases. Ware and colleagues (48) also suggested that this somewhat stronger association need not imply any special risk associated with maternal smoking. They argued that a more plausible interpretation is that children are more likely to be with their mothers than with their fathers at the times smoking occurs.

3.2.DAY CARE AS A CONFOUNDING VARIABLE

Reviewers have concluded that exposure to parental smoking increases the risk of upper and lower respiratory diseases in infants. Day care attendance also greatly increase the occurrence of upper respiratory infections and perhaps some lower respiratory illnesses in infants and toddlers. However, studies focusing primarily on the effects of passive smoking have failed to control for day care attendance and many of the studies on day care infections did not consider parental smoking as a possible confounding variable. Moreover, none of the studies in either area considered the smoking habits of day care workers.

Seven of the day care studies (14, 19, 30, 39, 45, 46, 47) did consider parental smoking. Two of these studies (14, 30) found a positive and independent effect for both day care attendance and maternal smoking. The effect of day care had the strongest in both cases. The remainder showed a statistical significance for day care attendance only. Pukander and colleagues (30) argued that the effect of day care attendance may have masked the effects of maternal smoking in these studies.

Given that almost 50% of mothers of children under 5 years of age

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now work outside the home, (28, 37) the smoking habits of private day care givers must be considered as an important variable in the development of childhood respiratory infections. (37). The Section on Allergy of the Canadian Pediatric Association (6) reported that infants admitted to hospitals for chest problems had been found to have significantly more day care givers who smoke than did control infants.

The smoking practices of workers in day care homes deserves immediate attention because this sector includes more children than the others and its popularity with mothers of infants and toddlers. Day care mothers who smoke and care for infant and toddlers probably spend as much time if not more with these children as their mothers. Thus, the smoking habits of these day care workers potentially confound the results of studies of the effect of parental smoking on the health of children.

4. RECOMMENDATIONS

4.1. REGULATIONS

Existing day care regulations clearly are deficient in mandating a safe and healthy day care environment. Federal regulation, while desirable, is not feasible at this time since the prevailing attitude today is away from federal intervention and toward state and personal responsibility. (49)

Before proposing solutions, Haskins (17) recommend that one should determine if the current state of day care regulation is causing the market to function improperly. He also warned that the scientific data necessary for a convincing case of market failure and an accurate assessment of benefits and costs are currently inadequate. Thus, the policy problems cannot receive a complete assessment until the state of knowledge improves. Meanwhile, he suggested that the most important interim steps are to give parents better information about day care and strengthen state regulations that are clearly related to health outcomes.

In designing health policies for day care, it is important to approach the problems comprehensively and constructively. (24) We must keep in mind the potentially positive as well as negative effects of substitute care on children's health (49, 50) and the reasons families turn to day care in the first place, namely, because both parents must have child care during work hours. (35, 50)

The regulation of day care homes, which contain the most children is an especially delicate issue. Bartlett and colleagues (2) warns that increased regulation of homes might have the effect of actually decreasing the availability of this mode of child care. Haskins (2) also cautioned that the sheer number of providers and the small size of these units would make effective oversight a

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formidable and expensive task.

Furthermore, if regulations are made too strict, costs will go up and day care centers will be put beyond the means of those who need them the most. The results might send day-care underground where it could operate without proper health supervision. (24)

Haskins (2) proposed the following steps to help parent get information:

1.Require homes and centers to permit unlimited and unscheduled visits by parents;

2.Put parents in a position of power and authority in the operation of day care through membership on boards;

3.Require all facilities to have written policies on health and that a copy be given to parents; and

4.Community Coordinated Child Care (4-C) be the primary actor in providing training and providing parents with information on child care.

The Symposium of Infectious Diseases in Child Day Care: Management and Prevention voiced unanimous support for strict adherence to a policy of frequent hand washing; and at least daily cleaning and sanitization of surfaces used for diapering, food preparation, and those that are mouthed by infants and toddlers. (1)

Fleming and colleagues (14) addressed the issue of passive smoking. They noted that the major difference between maternal smoking and day care attendance, is that maternal smoking is preventable, whereas day care attendance is not. Child care is an irreplaceable service which also results in the transmission of disease. Therefore, reduction of other risks among children who attend day care is the most practical approach.

4.2.RESEARCH

Little is known about the actual practices of licensing agencies and the effectiveness of regulation. Do children in day care visit health professional more often for infectious diseases? If they do, is it because day care programs are urging more frequent consultations, because the parents are more anxious about the threat that their children will be excluded from day care if sick, or because there actually are more infections. (1)

The sparse data available regarding family day care make it important to pay more attention to this mode. Since day care homes includes more children, particularly infants and toddlers, it is important to understand the disease experiences in these homes.

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Much more needs to be known about the impact on child health and the practice of family day care before this approach can be knowledgeably recommended. (2)

The smoking patterns of day care workers, particular those in day care homes, should be identified. Data from the National Health Interview Surveys, 1978-1980 put the prevalence of smoking among female child care workers at 28.9%. These data excluded private household child care workers with whom most children, particularly infants and toddlers, are cared.

4.3.EDUCATION

At the moment, Parents must judge for themselves the quality of care given to their children. However, most parents do not know what to look for in a day care setting and there are no federally sanctioned standards. (35, 50) There are, however, guidelines that are generally agreed upon within the child development community. (35) A checklist for quality of care has also been developed and found to differentiate between centers of high quality and low quality. The checklist includes one item on smoking: "Adults do not smoke in rooms where children are." (7, 35)

The body of information now available on the prevention and management of infectious diseases in day care should be widely disseminated to day care providers, public health workers, health care providers, and parents. Public health authorities must be armed with low-cost materials to distribute to day care providers. (1)

5. SUMMARY AND CONCLUSION

The children of working parents are receiving day care primarily in their own home, family day care homes, and day care centers. Family day care is the largest of the three sectors but day care centers represent the fastest growing segment. Studies, mostly in Scandinavian countries, have demonstrated that children attending day care centers and probably family day care have more respiratory infections than children receiving in-home care. The effect is greatest among infants and toddlers.

Another group of studies have linked parental smoking, primarily maternal smoking, with an increase in respiratory diseases among infants. However, most of these studies did not control for day care attendance. The few studies that controlled for parental smoking and day care attendance showed a consistent and positive association for day care attendance. The results were mixed for parental smoking. None of the studies, however, controlled for the possible confounding factor of exposure to environment tobacco smoke by children who were cared for by day care workers who smoked cigarettes.

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Future studies controlling for potential confounding factors are urgently needed in this area. The smoking practices of day care workers, particularly family day care homes, may have been a major uncontrolled confounder in past studies.

In the interim, parents should be alerted about not only the harmful effects of parental smoking on infants but the potential added exposure from day care workers. The children of nonsmoking parents who are provided day care by smokers could possibly have an exposure to environmental tobacco smoke at a rate similar to that of children with one or more smoking parent. Day care providers must also be educated about the possible compounded effect of passive smoking and day care attendance on the increased risk of respiratory diseases among infants and toddlers.

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FIGURES AND TABLES FOR CHAPTER 9

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7. TABLES

TYPE OF CHILD CARE BY AGE OF CHILD

Table 1

AGE	< 1 Year	1-2 Years	3-4 Years	TOTAL
IN-HOME CARE	34.3%	33.3%	24.6%	30.5%
DAYCARE HOME	42.7%	43.0%	35.4%	40.2%
GROUP CARE	5.3%	11.7%	25.8%	14.8%
MOTHER	9.2%	8.6%	9.9%	9.1%
TOTAL	91.5%	96.6%	95.7%	94.6%

Source: O'Connell and Rogers, 1982 (28)

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CARE GIVER BY AGE OF CHILD

Table 2

AGE	< 1 Year	1-2 Years	3-4 Years	TOTAL
FATHER	13.9%	15.8%	11.0%	13.9%
MOTHER	9.2%	8.6%	9.9%	9.1%
GRANDPARENT	22.4%	16.8%	14.6%	17.2%
OTHER RELATIVE	11.3%	12.3%	12.8%	12.1%
NONRELATIVE	29.4%	31.4%	21.6%	27.5%
GROUP CARE	5.3%	11.7%	25.8%	14.8%
TOTAL	91.5%	96.6%	95.7%	94.6%

Source: O'Connell and Rogers, 1982 (28)

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CAREGIVERS BY TYPE OF CHILD CARE AND AGE OF CHILD

Table 3

AGE	< 1 Year	1-2 Years	3-4 Years	TOTAL
IN-HOME CARE				
Father	13.9%	15.8%	11.0%	13.9%
Grandparent	8.9%	6.3%	3.6%	5.9%
Other Relative	5.1%	5.0%	5.7%	5.2%
Non-relative	6.4%	6.2%	4.3%	5.5%
DAY CARE HOME				
Grandparent	13.5%	10.5%	11.0%	11.3%
Other Relative	6.2%	7.3%	7.1%	6.9%
Non-relative	23.0%	25.2%	17.3%	22.0%
GROUP CARE				
Nursery	1.7%	3.2%	11.7%	5.6%
Day Care Center	3.6%	8.5%	14.1%	9.2%
MOTHER	9.2%	8.6%	9.9%	9.1%
TOTAL	91.5%	96.6%	95.7%	94.6%

Source: O'Connell and Rogers, 1982 (28)

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CHAPTER 10

NO SMOKING POLICIES AT THE WORKSITE

A Look at What Companies Are Doing Today

By Ruth Behrens

Washington Business Group on Health*

The number of businesses with smoking polices has burgeoned. A national, predictive survey released in 1987 by the Office of Disease Prevention and Health Promotion, U. S. Department of Health and Human Services, found that 27.2 percent of all U.S. companies with 50 or more employees have a formal smoking policy. Of these, 40.4 percent report the policy is in place to protect nonsmokers; 39.5 percent report the policy is designed to comply with regulations; 12.7 percent report a need to protect equipment; and 7.4 percent advise that the policy is designed to protect employees at high risk for health problems.[1] A more recent study, that looked only at large and medium-sized companies, the 42nd annual Northwestern University Lindquist-
Endicott Report, found that 70 percent have restricted, or are prepared to limit, smoking inthe workplace. The study was released in early 1988.[1a]

The development and implementation of a no smoking policy within a business is a multi-faceted process. Experiences of the growing number of companies that have developed written statements spelling out how smoking will be limited or prohibited illustrates vividly

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that the process involves many individuals and groups, and that deliberations often are emotionally charged.

This chapter contains a series of case studies outlining how several companies have successfully approached this process. But before discussing company-specific examples, there is merit in examining some of the key issues that must be looked at by any company considering developing a no smoking policy. Among the questions to be answered are:

- What kind of specific smoking restrictions are best for the company?
- What benefits can be realistically anticipated from the policy?
- How should employees be involved?
- How should unions be involved?
- What kind of education should be offered and to whom?
- What kind of incentives should be offered?
- How should the policy be enforced?

Further details about each of these steps are contained within the "Case Studies" section of this chapter.

Options for Smoking Restrictions

Restrictions on smoking in the worksite are not new.

For year--even decades--businesses have had policies that banned smoking in specific areas such as elevators, hallways, auditoriums,

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sections of cafeterias, laboratories, rooms with delicate equipments, etc. In many instances, these restrictions were imposed because of laws or ordinances requiring them or to protect property. Before the 1980s, they were seldom implemented for health reasons. The assumption was, of course, **the entire company is considered a Smoking Permitted area unless otherwise specified.**

Another type of policy began appearing with regularity in the mid and late 1980s. It **banned smoking throughout the company except in designated areas.** While many of these policies did not necessarily put greater limits on smoking--often allowing offices and work areas, special lounges, large parts of the cafeterias, etc, to be designated as Smoking Permitted areas, they did set the precedent that **the company is Smoke Free except in specified areas.**

While the difference between these two types of policies may seem subtle at first glance, there is a strikingly different corporate philosophy underlying the two approaches. And in the late 1980s, it was this latter approach--**establishing a smoke free company, possibly with a few, carefully selected areas that permit smoking**--that appeared to be setting the pattern for worksite smoking policies.

According to a spokesperson for Texas Instruments, Inc. (TI), determining what approach to take in limiting smoking was the most

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difficult aspect of developing and implementing its policy.

As TI and many other companies have found out, designating even a few smoking areas within a company can still pose serious health hazards for employees because smoke from lounges, cafeterias, hallways, and enclosed offices, gets into the ventilation system and is circulated throughout the building, including into no smoking sections. (See "Case Studies: Pacific Northwest Bell.") As an interim step in a phased-in nonsmoking work environment, Pacific Mutual Life Insurance Company, Newport Beach, California, installed electronic filters in the temporary smoking area of its cafeteria.

TI chose to avoid this problem by eliminating smoking from the worksite except for designated smoking areas which were, to the extent possible, separately ventilated. Similarly, the headquarters complex of General Telephone of California prohibited smoking in all areas except a small portion of the cafeteria that has its own ventilation system.

For others like American Family Insurance Group, Madison, Wisconsin, Pennsylvania Blue Shield, and UNUM Life Insurance Company, Portland, Maine, the choice to provide separate ventilation was either too expensive or physically impossible, so they chose to ban smoking completely at the worksite. On October 1, 1987, Ralston Purina's headquarters in St. Louis, Missouri, became the first Fortune 500 company to completely ban smoking in

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its facilities.

Clearly, the trend is moving toward more and more companies banning smoking or severely limiting it. Most require that all visitors abide by the company's regulations. Some will not allow smoking on company property, including grounds and parking lots. Groups like Michigan Bell, which has a large number of motor vehicles, are expanding their bans to all company-owned vehicles. However, others are voiding a ban in company cars and trucks because they believe enforcement will be virtually impossible.

A few companies have gone even further, and may be bellwethers for a future tend. These companies require that all new employees sign a statement that they are nonsmokers, even on their own time. Company **policies prohibiting the hiring of smokers** got nationwide publicity when Acoustical Products Company, a subsidiary of Chicago-based US Gypsum Corporation, announced that because of exposures to fibers that could have adverse health effects, all present workers were required to quit smoking or face termination, and in the future, only nonsmokers would be hired. The Non-Smokers Inn in Dallas, Texas, provides only nonsmoking rooms and hires only individuals who do not smoke. At Cardinal Industries, Columbus, Ohio, new employees must state on the application form whether or not they smoke, and only nonsmokers will be hired; but the company does not make any effort to check or test to find fibbers. Louisiana Pacific Corporation, a Portland, Oregon-based national

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timber company with 15,000 employees, does not hire smokers in any of its plants or in corporate offices "because of the medical costs, absenteeism, environment of smoke in the workplace, the fire problems in the mills, and lung cancer."[2]

The vast majority of companies still do not require that new employees be nonsmokers. But many companies with strict bans are seeing fewer smokers apply. "Why would a smoker want to work for us," one company spokesman said, "when we deprive him of his habit for eight hours every workday?"

Benefits of No Smoking Policies

Developing and implementing a worksite no smoking policy is not easy and may cause a great deal of discomfort for smokers and management alike. So why do companies do it? What benefits do they receive?

In a recent national survey of all types of worksites with 50 or more employees, the Office of Disease Prevention and Health Promotion, U. S. Department of Health and Human Services, asked those with smoking programs what benefits they perceived.

- 41 percent said smoking control policies and programs improved employees' health;
- 16 percent said they increased employees' productivity;
- 9 percent said they improved morale, and
- 8 percent said smoking control activities reduced costs.[1]

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Some companies have conducted evaluations of the results from their smoking control efforts. Several of these studies, along with some anecdotal findings, are reported in this chapter's Appendix, "The Economic Justification for No Smoking Policies."

To many companies, a reduction in the number or percent of employees who smoke is benefit enough from a policy. Smokers dropped from 21 percent of the workforce to 16 percent in two years at UNUM Life Insurance Company. In addition, 87 percent of the smokers reported they were smoking less after the policy was implemented.[3] At Pacific Northwest Bell, smokers dropped from 28 percent at the time the ban was implemented to just 20 percent of employees two years later.

Employee Involvement

Some companies, especially those wanting quick results that can be controlled, develop smoking policies at the top executive or management level and announce them to the employees. But a more frequently seen pattenr today involves employees in the process of formulating and implementing a policy from the outset--but with varying degrees of direction from management.

In some companies, the involvement takes the form of responding to a charge, such as: examine the issues and problems related to smoking at our worksite and present your recommendations for a

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policy and implementation plan to deal with them to management in three months. In others, management may decide that smoking is a serious health hazard to its employees and that smoking is to be eliminated in 12 months. This organization's charge to employees might be to: review how other companies have successfully moved to a smoke free workplace and present your recommendations for steps our company should undertake during the next 12 months to make that transition both smooth and as painless for smokers as possible.

But if employees are to be involved, it is important that their contributions have meaning and be listeneded to objectively by management.

When tracing the history of smoking policies in organizations, it is not unusual to find that the initial push to limit or eliminate smoking came not from management, but from the employees, themselves.

At UNUM Life Insurance Company, employees' complaints, coupled with a Maine law requiring employers to reduce smoking, resulted in the company-wide ban. Pacific Northwest Bell emphasizes that no company officer or executive advocated its move to implement a smoking policy. Rather, the impetus came from employees. A grass roots group conducted a survey of workers and eventually recommended that PNW Bell ban smoking. The Employee Advisory

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Council at Cardinal Industries' Sanford, Florida, plant initiated the idea of a tough no smoking stand. (See "Case Studies--Cardinal Industries and Pacific Northwest Bell")

At Holiday Corporation, Memphis, Tennessee, a task force of employees developed a Clean Air Policy covering its headquarters offices. The task force was originally set up as a Wellness Committee a full year before work began on the smoking policy. The employee group researched various aspects of the smoking problem by gathering research, talking to other companies that had already done it, and working with the local cancer society and lung association. A survey was conducted of all employees to identify their habits and attitudes related to smoking. The Task Force, itself, created the phased-in process that resulted in Holiday Corporation headquarters and several of its subsidiary groups going smoke free January 1, 1987.

But the act of involving employees is not always as easy as it might seem, according to Charles Nielson of Texas Instruments. It is important to involve employees in the process of developing a policy as early as possible, and a survey of their habits and attitudes provides invaluable data to management, says Nielson. But because TI has so many locations, timing of an employee survey was sometimes very difficult; in some locations, the policy had already been set by corporate headquarters before the attitude survey could be conducted. As a result, some employees felt they

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were being manipulated. "Data is vital to planning, but timing is also important so that the company maintains its credibility," cautions Nielson.

Union Involvement

In any unionized organization, consideration must be given at the outset to how and when unions will be involved. Popular thinking just a few years ago was that unions would block most company-sponsored wellness efforts, particularly those that interfered with personal freedoms, such as smoking. But through the work of several pioneering unions such as the Amalgamated Clothing and Textile Workers Union, the United Steelworkers Union, and the United Auto Workers, as well as the efforts of national groups including the Workplace Health Fund, more and more labor groups as willing to cooperate with management in reducing smoking if they are approached properly--and early in the process.[4]

Unions also recognize that their membership reflects closely the national averages, therefore the vast majority of their members do not smoke. As a result, many unions are receiving increased pressure from their membership to help control smoking in the workplace. After having been involved in all aspects of policy development, the Communications Workers of America sent a memo to its 8,000 members at Pacific Northwest Bell acknowledging that the company was implementing a smoking ban, but stating that CWA would not oppose it because of the possibility that nonsmoking members

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would sue the union--and probably win. (See "Case Studies: Pacific Northwest Bell.")

In late 1985, the Workplace Health Fund, in cooperation with the Office of Disease Prevention and Health Promotion, (US DHHS), held a conference of union people to discuss the merits and value of health promotion. One of the outcomes of the meeting was a set of criteria for union involvement in worksite wellness efforts.

Among the recommendations were two that placed heavy emphasis on the need to have a good working relationship between the union and management before attempting to implement any kind of wellness program. "Worksites in which labor and management are not cooperating to bring health and safety hazards under control should not be sites for health promotion activities." In addition, "where the worksite is not under control or the employer is uncooperative, and where the union has established the need for health promotion, the programs should be conducted outside the worksite."[4]

But for any unionized company considering a smoking policy, the first step must be to look carefully at its union contracts, particularly for any wording that might guarantee members the right to smoke. If such agreements exist, the likelihood of the union supporting a no smoking policy is slim.

Sue Pisha, area director of the northwest region of the

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Communication Workers of America, believes that with motivational information and education, there is the potential for unions to eventually become a proactive force for nonsmoking policies. "Policies seem to eliminate in-fighting," she says. "Without a policy, the issue is messy and polarizing."[5]

Education

A companion element of virtually every successful workplace no smoking policy is an educational program designed to inform employees about the new rules and to provide opportunities for smokers to kick the habit. While behavior modification programs are the most commonly presented, some companies have offered innovative approaches such as acupuncture, hypnosis, aversion therapy, self-help materials, hot lines, and multi-day intensive programs for hard-core smokers. In addition to on-site opportunities, businesses have gotten good results by encouraging participation in community-sponsored stop smoking classes merely by providing lists of sessions available through reputable groups such as cancer and lung associations, hospitals, Ys, and for-profit organizations.

Because quitting can be very difficult and often is greatly enhanced by peer and family support, many companies make cessation opportunities available to spouses and other immediate family members, as well. (See "Incentives.")

UNUM Life Insurance Company offers classes for a usually over-

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looked group, nonsmokers. The classes are designed to help those who do not smoke understand the problems faced by smokers trying to quit and to urge them to encourage fellow workers to quit or to refrain from smoking. At Rainier Bank, Seattle, Washington, stress management classes were offered to help smokers adjust to the policy as well as to assist those who were trying to quit.

While there are not national data available on worksite quit rates, strong worksite programs claim anywhere from 20 to 50 percent quite rates after one year. However, most published studies report six-month abstinence rates of 30 percent or less.[6] As a result of an intensive smoking cessation campaign, Johnson & Johnson, New Brunswick, New Jersey, reports a two-year success rate of 23 percent **of all smokers in the company**, not just 23 percent of those who went through a program or completed it.[7]

Incentives

Many companies go a step beyond offering cessation classes by providing incentives for smokers to quit. The most widely used incentives for smokers are monetary, often tied to completing a cessation program and/or stopping smoking. Many companies offer cessation classes free to employees and their families, often during company time, or reimburse them for the cost of taking a community-based class. Others, like the Utah State Department of Health, reward smokers who actually quit. The "Healthy Utah" programs pays smokers who quit $25 at the end of three smoke-free

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months, another $25 after six months, and $50 at the end of a year of not smoking.[8]

Nonmonetary incentives, too, can be appealing. Employees who participated in a 24-hour "Cold Turkey" stop smoking day at MSI Insurance, Arden Hills, Minnesota, became eligible for a drawing for a frozen turkey. Those who quit for six months were eligible for a drawing for a free YMCA membership, and anyone who stayed off cigarettes for a full year was eligible for a weekend vacation.[10]

Some companies have gotten creative in finding ways to reward employees who are nonsmokers or who quit before a policy goes into effect. Employees who take a health risk appraisal at Westlake Community Hospital, Melrose Park, Illinois, receive a $50 "bounty" for participating plus several "good health bonuses" including $25 for not smoking. Weekly paychecks at Speedcall Corporation, Hayward, California, include an extra $7.00 for those who do not smoke at work. Backsliders who light up one week and lose their reward are encouraged to get back quickly to not smoking; so the next week without smoking earns the $7.00 bonus again.[8]

Businesses also offer nonsmokers discounts on life and health insurance, a very visible and tangible incentive to stop smoking and improve health.

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Smoking Policy Enforcement

Without a doubt, one of the most difficult questions asked by companies considering a smoking policy is "How can a no smoking policy be enforced?" The response from most businesses that have moved to a ban is that the company must first demonstrate to employees that it is serious about eliminating smoking in all or parts of the building. Second, it must handle violations in the same way that infractions of all other personnel policies are dealt with.

Cardinal Industries had a highly visible and dramatic way of demonstrating its commitment. Its president, Austin Gurlinger, a cigar smoker, stated to all employees that he would refrain from smoking at the workplace.

Most companies say that no people have quit their jobs because of the new rules. However, most also point out that employees have "tested" the policy, with some pushing it all the way to probation. According to Dick Becker, employee services representative for American Family Insurance Group, "Some employees tested the waters, sneaking cigarettes in the restrooms. Supervisors let it be known that smoking would be treated like any other violation of policy, for example, inappropriate dress."[3] Holiday Corporation follows its usual procedure for any company rule--first a verbal warning, then a written warning, followed by a "final" warning, and if

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necessary, termination.

But all agree, termination is not the objective. Everything possible should be done to encourage employees to comply, and most feel that peer pressure is the best policing mechanism. However, when an employee continues to break the rules, he or she must be disciplined appropriately, or the entire policy will crumble. (See "Case Studies.")

CASE STUDIES

Following are case studies illustrating how four widely different companies approached the development and implementation of a policy to reduce or eliminate smoking within their organizations.

CARDINAL INDUSTRIES, INC.

A total ban on smoking on any company property exists; all new employees must attest to being nonsmokers.

Beginning January 1, 1987, the 8,650 employees of Cardinal Industries were assured of a totally smoke free work environment.

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One year prior to the ban, Cardinal, the nation's largest manufacturer of modular homes, had taken an even more dramatic step by instituting a multi-faceted policy that included hiring only nonsmokers as new employees.

Benefits Anticipated

Although insurance carriers are saying it will take 12 to 18 months to see any decrease in insurance rates, Cardinal's management expects to significantly lower operating costs, increase productivity, reduce absenteeism, and eventually pay lower insurance premiums as a result of the new policy. Even more importantly, it expects to improve the health of its key asset--its human resources.

But employees at Cardinal's Sanford, Florida, location--one of four regional sites throughout the country--are convinced they would have gone smoke free even without the corporate edict. Why? Because employees wanted it, and because management recognized the negative impact of smoking on employees' health and productivity. The passage of Florida's Clean Indoor Air Act in October, 1985, focused attention on the plant's efforts and established it as one of the most progressive worksite no-smoking policies in the state, stimulating a letter of commendation from the governor.

Employee Involvement

Because of the nature of materials used at Cardinal, the second

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largest residential builder in the country, the company had a long-standing policy prohibiting smoking in its five manufacturing plants. But at the Sanford location, the real push for a tough policy that extended beyond the manufacturing facility came through its Employee Advisory Council in late 1985. Made up of employees elected by the workers within each department, the Council meets regularly with top management.

Based on employees' suggestions, a three-phase policy was developed and implemented Jan. 1, 1986, that gradually eliminated smoking in meeting rooms, the cafeteria, and other common areas during the year. As part of the policy, which was designed to make Cardinal smoke-free by the end of the year, only non-smoking personnel were hired. Current employees' smoking privileges (in designated areas) were grandfathered for the remainder of the year.

But before the policy was implemented, it required approval by top management, including the company's 33 year-old founder and president who was a cigar smoker--a situation that has stopped many other companies with good intentions. "We had been looking for ways to reduce our health care costs and at the same time improve efficiency and productivity," said a company spokesperson, "and the evidence about the health consequences of smoking were too powerful to ignore. When you add the fact that Cardinal pays for 100% of employees' health insurance, the decision seemed inevitable."

Enforcement

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In many ways, the fact that the chief executive at Sanford was a smoker aided in convincing employees that the plant was serious. The announcement that only nonsmokers would be hired and that there would be no exceptions to the rule--even the president--helped overcome one of company's biggest obstacles to successful implementation...convincing employees that the company is serious about the ban.

A second advantage Cardinal has going for it in terms of enforcement is a highly desirable work environment. It pays top benefits and offers excellent working conditions. An employee must balance sacrificing his/her smoking habit for eight hours each day with sacrificing a job at Cardinal. So far, Cardinal has won every time. Not only has no one quit, but the ban has not even been tested. "They know we are serious, and if they test us, they must be willing to live with the consequences." Management also believes that the long-standing positive environment among employees and management has contributed to the easy transition.

Education

During the 12 months between the announcement and the implementation, various company-paid educational programs and cessation classes were offered. In addition to regular stop smoking seminars provided after business hours, employees and their families also were offered a hypnosis program, and for those who felt they were addicted to smoking, an intensive two-day, off-site

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treatment program was provided. FDA approved pharmaceuticals also were offered as quitting aides. During the period preceding the ban, smoking areas within the locations were gradually restricted until, on January 1, 1987, the entire company became smoke free.

Blue Collar Workers

Although no survey has been taken to determine how many employees have quit smoking, a survey taken before the ban was implemented revealed that more white collar employees than blue collar workers were smokers. At the Florida location, for example, some 40 to 45 percent of employees could be classified as "blue collar." But partly because the manufacturing plants were always nonsmoking, there has been no particular problem in implementation among the laborers.

Off-Job Smoking

In a Position Paper discussing its policy, Cardinal Industries states, "The program only concerns itself with smoking in the workplace and not what employees do on their own personal time. Cardinal Industries never has, and never will try to regulate the activities of its employees on their own personal time." Thus, while Cardinal's application form asks prospective employees whether or not they smoke, and while its policy prohibits the hiring of smokers, no attempt is made to test employees or to check on their off-work habits.

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TEXAS INSTRUMENTS

Smoking is prohibited in all owned and leased facilities except in specific locations in each facility that are designated as smoking areas and, to the extent possible, are separately ventilated.

In late 1985 and early 1986, several of the 37 major sites of Texas Instruments began implementing their own smoking policies as a result of employee complaints and local Clean Air legislation. Rather than be faced with 37 different policies to implement, TI made a decision to implement a single corporate-wide policy.

Employee Habits and Attitudes

Before embarking on policy development, TI surveyed its employees to learn how many smoked and how they viewed worksite smoking restrictions. About half of the more than 50,000 workers surveyed took the time to respond, revealing that 77 percent of TI employees were nonsmokers or exsmokers, and that just 23 percent were current smokers. Of those who smoked, over 40 percent said they wanted to quit.

Corporate Objectives and Policy

Before designing the Clean Air Policy, top corporate management agreed on three objectives that would form its underlying

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philosophy. There were to:

- o provide a healthful and safe working environment;
- o ensure high quality in all TI products; and
- o initiate the company's clean air approach rather than be forced to react to legislation (including the possibility of legislation from many different states and municipalities).

From these objectives grew TI's Clean Air Policy.

"It is the goal of Texas Instruments to provide for its employees a healthful and safe working environment. In accord with this goal, Texas Instruments will prohibit smoking in all TI owned and leased facilities, except for specific locations in each facility which are designated as smoking areas."

Education and Training

To underscore the importance of the new policy, the eight-month, phased-in implementation process took a top-down track, with the President and CEO Jerry Junkins working directly with a key operating manager and the personnel director from each location throughout the organization. During the session, Mr. Junkins emphasized the organization's complete commitment to the new personnel policy and each individual manager's responsibility for its successful implementation. These teams then headed up similar training programs in their own locations. Training sessions were

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conducted for selected managers using a centrally prepared manual to ensure consistency among the 37 locations. Specially developed brief video tapes offered all employees an introduction to the policy (3 minutes), briefed managers and supervisors on issues related to smoking (10 minutes), and assisted managers and supervisors in learning techniques for resolving smoking-related problems at the worksite (16 minutes).

Making every effort to assist smoking employees to prepare for the new policy, TI provided company-paid smoking cessation programs on company time during the initial phase-in of the clean air program. Classes were scheduled to accommodate workers on all three shifts, and "maintenance sessions" were offered to provide additional support.

More than 4,700 employees signed up for cessation classes, representing 40 percent of the company's smokers--almost exactly the percent that said they wanted to quit in the employee survey. Of the group, 3,235 completed all the required classes (including maintenance classes), with 1887, or 58 percent, reporting they had quit by the end of the program.

As a further aid, a Tip Sheet, "How to Make Life Easier Until the Next Cigarette Break" provided "some practical suggestions to help you when you need to change your regular smoking routine." A "Wrap Sheet: Daily Cigarette Count," designed to be wrapped around a pack

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of cigarettes, offered an easy place to keep track of how much was smoked, when and why, in the hope that the information would assist the smoker in altering his/her habits.

However, all communication was not downward! Employees were given opportunities to ask questions and voice concerns during educational programs. Special attention was paid to employees concerns and complaints in in-house communication vehicles, as well.

Facilities Modification

Because TI chose to designate a limited number of areas in each building as smoking areas rather than to completely ban tobacco, it faced the problems of recirculating contaminated air. Thus, where necessary and possible, facilities were modified to provide separate ventilation. In addition, all cigarette machines were removed from TI facilities and a decision was made that no new ones would be installed.

Enforcement

TI made it clear from the beginning that a new personnel policy had been established that would be monitored and enforced in the same way as all other policies, such as attendance. Thus, anyone found smoking in non-designated areas would be given an oral warning. If there were no further problems, no further action would be taken. However, with subsequent smoking incidents, the

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employee would be given written guidance, followed by probation for additional infractions, with termination as a final step. But TI stressed to all supervisors that they should make every effort to educate smokers about the importance of the policy, rather than to be heavy-handed. After nine months, "two or three cases" have gone to probation, but no one has been terminated because of smoking. Considering that 50,000 to 60,000 employees are covered by the policy, this is an excellent record, says Charles Nielson, Vice President and Manager of U.S. Employee Relations.

Advice: Keep Policy's Purpose in Perspective

Nielson cautions other companies considering establishing a no smoking policy that one of the most difficult problems they face will be keeping the desire to eliminate the health hazards of smoking at the worksite in proper perspective.

TI made a corporate decision to eliminate smoking at the worksite except in designated areas. The decision was a business decision, not a moral or a value judgement. TI, which has 50 percent of its business in semiconductors, is facing intense competition, according to Nielson. Therefore, it must have productive employees. And that means it must have good relationships with all its employees. But smoking is an emotional issue for many people, both smokers and nonsmokers. "I'm not sure those of us in the personnel field have yet learned how to deal with this kind of highly charged issue and still maintain our productivity," states

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Nielson. "It takes a lot of hard work to achieve the desired atmosphere of teamwork, rather than an adversarial relationship."

RAINIER BANK/RAINIER BANCORPORATION

Following a one year phase in period, smoking is banned in all 200 domestic facilities and in car pool vehicles.

In September, 1985, the 5,800 employees of Rainier Bancorporation's U.S. facilities received a communication from their President, John D. Mangels stating that "We are committed to insuring a healthful and comfortable environment for all employees." As part of that commitment, he announced, the corporation would become smoke-free on October 1, 1986. As part of a transition plan, beginning October 15, 1985, smoking would be restricted to designated areas, and the company would sponsor and pay for smoking cessation classes to assist employees who choose to quit.

Rainier Bancorporation is headquartered in Seattle, Washington, with 200 offices in Washington, plus Alaska, Oregon, California, Hawaii, Arizona, New York, and the Far East.

<u>Health Threats, Employee Complaints, Legal Concerns Prompt Policy</u>

According to Peter Broffman, personnel officer for Rainier Bank,

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the major subsidiary of Rainier Bancorporation, the policy resulted from three converging issues, the major one being a concern for employee health and wellness. Additional factrors were an increasing number of employee complaints about smoke in the workplace, and the changing legal and regulatory climate. In July, 1985, the state of Washington had adopted a Clean Indoor Air Act that prohibited smoking in public places, including public areas of banks, and there was reason to believe that unless employees acted on their own initiative, there might also be legislation regarding privaate workspace. That, coupled with an increasing number of court cases upholding the right of employees to have a smoke free workplace, added impetus to the development of a policy.

Communications Vital to Implementation

Once the decision was made to go forward with a phased-in ban, communications with employees became a key link to successful implementation. Emphasis was placed on the fact that Rainier was prohibiting **smoking** at the workplace, not smokers.

Phase-In Period

During the transition period, managers were given discretion to determine the most appropriate way to make the transition. The company policy stated that "The needs and 'comfort level' of both smokers and non-smokers should be considered during this period." Guidelines for Phase I stated, in part:

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- All common areas, including lobbies, elevators, conference rooms, hallways, libraries, rest rooms and computer rooms will be smoke free.
- In open-office work environments, managers should use discretion in deciding whether those areas should be smoke free. Individual employees may, of course, designate their assigned immediate work space as a no-smoking area.
- Employees with enclosed offices may designate their area as a smoking or no-smoking area. However, the rights of non-smokers who must come into an enclosed office to conduct business should be respected.
- Lunchroom and lounge areas will be divided into areas for smokers and non-smokers. Managers are given discretion to divide the rooms as appropriate for their locale.

According to Broffman, there were relatively few difficulties in the initial phase of implementation. The few problems that did exist were due largely to differences in the ways various managers chose to implement and police their smoking restrictions. Occasionally disputes arose over what areas should be smoking and nonsmoking, especialy in the smaller branch offices where there were few options for allocating space. For the most part, Broffman says, the problems were minor and easily resolved when the total prohibition was enforced.

However, in retrospect Broffman believes that a shorter transition

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period might have been more desirable. "A three to six month phase-in period probably would have been adequate," he says. "Many smokers go through a adjustment period. A few indicated that putting off the inevitable ban for too long really isn't doing them a favor because it prolongs the period of anxiety and allows them to procrastinate in making the adjustment. Also, a shorter transtition emphasizes the resolve of the company to become smoke free." In addition, any employees who want to defeat the policy will use the entire phase-in period to rally support. A shorter transition period would shorten the debate and lessen the possibility that the detractors will succeed.

Enforcing the Ban

Phase II, the total smoking ban, was introduced in a low-key, matter-of-fact manner: a simple "reminder" that smoking would be prohibited in all work areas. With the exception of minor, final protests by a few "die-hards", employees accepted the new policy. Rainier has received no formal complaints, has had no problem with recruiting, and no one has resigned. The only complaint Broffman is aware of is that a few employees who still smoke do so immediately outside company building during breaks, and some employees are concerned about the impression this gives to customers entering the bank.

Because of its stance that Rainier is eliminating smoke, not smokers, the organization makes no attempt to discriminate against

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hiring smokers.

Biggest Obstacle to Policy: Fear

Broffman acknowledges that when the policy was first proposed, there was concernon the part of a few senior manager of "What might happe." Although the majority and the leadership of senior management supported the policy, a few were initially concerned that there could be mass defections, that disagreements about smoking would cause major disruptions in work units, and that it courl turn into an "employee rights" issue. However, these things did not happen at Rainier.

"We had more complaints from nonsmokers before the policy was implemented than we got from smokers after it was enforced." Broffman says. His advice to other companies considering a smoking ban? "Do it! You can make it work!"

PACIFIC NORTHWEST BELL

Because of health concerns related to smoke, PNB does not allow smoking in any company facility.

On October 15, 1985, Pacific Northwest Bell became the first large company (15,000 employees in 750 buildings) to go completely smoke free. Its policy is simple:

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"To protect the health of PNB employees, there will be no smoking in any company facility."

Options for Smoking Restrictions

Prior to the establishment of the policy, PNB had allowed each work group to decide, itself, whether or not it would be a smoking area. Problems arose, however, when adjacent work groups had differing approaches. Smoke would drift around barriers, waft across no-smoking desks, and generally infiltrate all areas of the building. Smokers assigned to no smoking areas would merely walk into work groups that permitted smoke, making the atmosphere even worse for nonsmokers in the area. Difficulties occurred even within individual units that voted to eliminate smoking. If 60 percent voted to be a clean air area and 40 percent voted for smoking, the question arose as to whether the wishes of four-out-of-ten employees could really be ignored.

While this kind of democratic approach had initially sounded like an easy way to avoid forcing a company-wide policy, it was seen as unfair and inequitable by most workers. No one was really satisfied and all the underlying problems still existed. Eventually both managers and employees began exerting pressure on PNB to develop a company-wide policy.

Employee Involvement

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In January, 1983, a Smoking Issues Steering Committee was established consisting of smokers, nonsmokers, and a group often forgotten, former smokers. Employees representing their unions and from the legal, health services, safety, and many operating departments were part of the task force. One of its first undertakings, an employee survey, brought an astonishing 74 percent response rate, attesting to the importance of the issue among workers. In addition to comments from those who were randomly surveyed, 151 people who were not part of the survey group made the effort to get copies from their friends so they, too, could have their view heard. They included 135 nonsmokers and 16 smokers.

Results of the 1983 questionnaire showed that 28 percent of PNB employees smoked, but that the majority of employees were bothered at least occasionally by smoke at the workplace, and almost 80 percent said the company should be concerned about smoking at the worksite.

Two-and-one-half years after its inception--following a great deal of research and discussion by the task force, as well as involvement in the issue by numerous Quality of Work Life teams and various ad hoc groups--the employee committee recommended to the officers that smoking be eliminated at PNB.

Union Involvement

At PNB, unions were instrumental in all phases of policy

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development. Not only were they included in the employee committee making recommendations about a future policy, but leaders of both unions were part of the June 1985 presentation to the company president of the committee's recommendation. "They were not there as advocates for a no smoking worksite," cautions Len Beil, director of human resources planning and employee involvement. "They were present, rather, to state that they had been involved in the process and what their positions would be on a strong policy. While they did not endorse the complete elimination of smoking in all buildings, they stated that their unions would not formally fight its implementation, either."

Beil adds that the the union members on the committee were "extremely helpful" in all aspects of policy development, and that while they never fought against the policy, they negotiated successfully for several compromises that proved to be fair and beneficial for all employees. Initially, the company wanted to reimburse employees for smoking cessation classes after successful completion. The union position was that PNB's goal was to assist and encourage employees to live with the policy and comply with it--not necessary to get them to stop smoking. Therefore, they pressed, the company should reimburse totally for cessation classes, whether or not the employee completed the series. On the issue of smoking in company vehicles, union representatives stressed the difficulty of enforcement and potential problems if cigarette butts were found in a company car or truck ash tray. On

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both issues, PNB went with the unions' requests. All employees got full reimbursement for taking a cessation class and smoking in company vehicles is a matter of "common courtesy." The unions also urged that any policy be consistent throughout all company locations and for all employees.

Education

The day the policy was announced, which was 90 days before the policy was to go in effect, PNB provided two telephone hot lines to answer questions about the policy and provide information on free cessation programs for employees and their dependents. (BNA Where There's Smoke: Problems and Policies Concerning Smoking in the Workplace. Washington, DC) A wide range of quit opportunities were made available, many on company time, with PNB paying all fees following completion. But PNB also garnered kudos from many employees by allowing them to take classes outside the company and still get reimbursement. The ability to choose their own quit method seemed to add to their commitment to succeed and helped encourage a friendlier attitude toward the policy.

Benefits

Within the first two years, 1,738 people had gone through cessation programs--1,353 of them employees, 360 spouses, and 25 dependents--receiving full reimbursement from the company for a cost of about $250,000. Is this investment worth it to PNB? "Yes," says Beil.

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"It is money well spent. This equals the cost of just two or three cancers cases. And we would much rather pay for 1,738 to try to quit smoking than pay the results of their continued habit."

Enforcement

PNB reports that there have been "no real problems" with enforcement. On the first day, there were reports that one or two people were smoking behind closed doors in several locations. But "word got around" and by the second day they were abiding by the rules. Although several people threatened to contact lawyers and a few employees tried to organize a Smokers Rights day, nothing significant came from any of the attempts to block implementation.

The bottom line: After two years, no one has quit because of the no smoking policy, there have been no grievances, and smokers at PNB have dropped from 28 percent to 20 percent in the two years since implementation. All in all, the company views its no smoking policy as an unqualified success.

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APPENDIX TO CHAPTER 10

ECONOMIC JUSTIFICATION FOR NO SMOKING POLICIES AT THE WORKSITE

By Ruth Behrens

Washington Business Group on Health*

The health hazards of smoking--including smoking at the workplace--have been well documented. Smoking greatly increases an individual's chances of contracting serious illnesses, such as cancer, chronic bronchitis, emphysema, and coronary heart disease, and of dying prematurely as a result of these diseases.

There is little doubt that smoking also has a significant economic impact. And because businesses pay a sizeable portion of America's total health care costs--estimated at $108 billion per year by the Employee Benefit Research Institute--they are paying much of the bill related to smoking through insurance premiums for their own employees, dependents, and retirees who smoke or breathe second-hand smoke, as well as for others through programs supported by state and local taxes. In other words, smoking is costing businesses a lot of money.

How much does smoking cost U.S. businesses? No one knows exactly. But a growing list of researchers are tackling the difficult job of attempting to identify these costs.

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Costs of Smoking to the Nation

Three major studies have addressed the question of what smoking is costing the nation.

In 1978, Luce and Schweitzer estimated the economic costs of smoking in the United States to be $47.6 billion. They further broke this down to $811 per adult smoker, or $1.56 per pack of cigarettes sold.[1]

In 1985, the Office of Technology Assessment, U.S. Congress (OTA), estimated that smoking costs the nation about $65 billion per year in lost productivity and health care costs alone. OTA estimates that smoking-caused illness results in $43 billion in lost productivity annually (or $1.45 for each pack of cigarettes sold), expenses borne largely by employers. Businesses also pay a significant portion of another $22 billion in smoking-related health care costs, since nearly two-thirds of the costs are incurred by those under 65. According to the OTA, combined lost productivity and health costs related to smoking equal $2.17 per pack of cigarettes sold.[2]

Most recently in 1986, a group of researchers, which included the former director of the government's National Center for Health Statistics, concluded that smoking costs the United States at least $53.7 billion each year in direct medical costs and salary losses

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alone. These cost estimates were calculated by comparing the health costs and income losses from smokers in excess of the same amounts incurred by nonsmokers. The study concluded that smokers are sicker and require more medical care than nonsmokers.[3]

The components of the $53.7 price tag were broken out as follows.

- Direct medical costs such as doctor bills, drugs, and hospital and nursing home expenses were $23.3 billion more for smokers than the average of nonsmokers.
- A total of nearly $9.3 billion was lost in salaries due to smokers being sick with smoking-related diseases including lung cancer, heart attacks, stroke, emphysema, and other respiratory illnesses.
- In 1984, lifetime earning losses from smoking related deaths were approximately $21.1 billion.

The authors characterize their findings as "conservative" since they "did not take into account the adverse effects of passive smoking, risks of abortions, stillbirths, and neonatal deaths, or deaths under age 20 that might be associated with smoking."

In their paper published in The Milbank Quarterly, Rice et al translated all three of these studies to 1984 dollars. The result is three analyses of the economic impact of smoking on the nation that demonstrate enough similarity to underscore that smoking does,

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indeed cost our country a staggering amount:

- Luce & Schweitzer show a cost to the nation of $52.8 billion per year in 1984 dollars;
- OTA, $62.2 billion in 1984 dollars; and
- Rice et al, $53.7 billion in 1984 dollars.[3]

Differing Methodologies Make Pinpointing Worksite Costs Hard

A number of researchers also have attempted to assess the specific costs of smoking to businesses. But many problems arise when attempting to identify one, or even a "best" methodology for arriving at these costs.

Among the difficulties in conducting any study of the costs of smoking is the fact that smokers differ from nonsmokers in several genetic, social, and economic characteristics that may contribute to disease. For example, the prevalence of smoking varies by race (more blacks smoke that whites), education (fewer college graduates smoke than persons with only some high school), income (males with lower income smoke more, while the opposite holds for women), and occupation (blue collar workers smoke more than professional or technical workers). If factors known to be related to health status and smoking habits are not controlled, the impact of smoking on health and the costs of smoking may be overstated.[3]

When examining smoking in the worksite, specifically, other methodologic issues must be resolved. Marvin M. Kristein, a

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professor in the departments of economics and community preventive medicine at State University of New York at Stony Brook, and William Weis, chairman of the accounting department, Albers School of Business, University of Washington, and a member of the board of the Smoking Policy Institute, Seattle, both published studies in the early 1980s identifying the cost to business of each smoking employee. Kristein estimated the cost in 1980 dollars to be between $336 to $601 per smoker annually,[4] while Weis placed the figure nearer $4500.[5]

Much of this rather staggering discrepancy is attributable to their selection of different categories of costs to include in the equation, the weight given each category, and the salary assigned to the average smoker.

In a more recent article (Nov. 1984), Kristein has calculated that the costs to business are at least $300 to $350 per average smoker per year for short term losses, alone--which excludes health care costs. Kristein identifies these short-term costs as fire, accidents, ventilation, cleaning, productivity, and occupational health risks.[6] Obviously, the addition of long-term costs such as medical care costs, absenteeism, early death, etc. would increase the total significantly.

According to Weis, business costs in at least ten areas are affected by smoking or smoking controls, including no smoking

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policies: health insurance; incremental absenteeism; life and disability insurance; fire, liability and industrial accident insurance; ventilation and energy consumption for heating and air conditioning; legal liability; property damage, depreciation and maintenance; time lost to the smoking ritual, employee morale, and corporate image.[7]

To help illustrate the differences between Kristein and Weis's total smoking-related costs, one can look at how each calculates the costs of absenteeism to employers due to smoking.

Weis uses government data that shows a smoker is absent 2.2 days per year more than a nonsmoker. Using $30,000 per employee as the average annual wage and salary, including fringe and payroll taxes, the company pays approximately $120 per working day for every employee on the payroll. Assuming a 25 percent return on payroll dollars, the direct cost to the employer is $150 per absence, excluding the cost of temporary replacements. According to this formula, the total cost per smoker per year due to absenteeism is $310. A similar system is used by Weis in determining costs in other categories.[7]

Kristein, on the other hand, used 1979 data showing smokers are absent 33 to 45 percent more than nonsmokers, or 2.0 days more per year, and assigns a daily salary of just $40 per smoker due to smoking (versus $150 for Weis). Thus Kristein includes from $40

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to $80 per smoker per year attributable to absenteeism in his total (versus $310 for Weis).[2]

While Kristein's estimates are based on what he called "real numbers" drawn from insurance companies, U.S. government statistics, and detailed academic studies, he cautions, "We lack meaningful 'case controlled' company comparisons of experience with smoking employees versus nonsmoking employees....In general, the emphasis is on underestimating the costs to business."[8]

Economic Impact of Smokers on the Worksite

Evidence also shows that, in addition to excess absences of two or more days per year, smokers exert other types of economic impacts on businesses over their nonsmoking counterparts.

- smokers have twice as many job related accidents as nonsmokers.[9]
- Smokers are 50 percent more likely to be hospitalized than those who do not smoke.
- Employers have been held legally responsible for at least part of the disability cost for smoking employees who contracted smoking related illnesses, in addition to claims from nonsmoking employees who were adversely affected by the smoke of others.[10]
- Companies with certain occupational hazards can expect greatly increased costs related to smoking. For example, an

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asbestos worker who smokes is ten times more likely to die prematurely than his nonsmoking coworkers. A smoking uranium miner has six times the risk of contracting lung cancer as a nonsmoker in the same job.[4]

In addition, many health consequences of smoking translate directly into increased health care costs, since employers pay for a major portion of these costs for their employees, dependents, and retirees.

- Heavy smokers (two or more packs a day) are 15 to 25 times more likely to die of lung cancer than nonsmokers, and overall, smokers are ten times more likely to die of lung cancer than nonsmokers.[11]
- Eighty to 90 percent of such long term severe lung diseases as emphysema and chronic bronchitis are related to smoking.[12]
- It is estimated that 30 percent of all cancers are caused by smoking. That means that 138,000 Americans died of cancer in 1986 because of smoking.[12]
- Heavy smokers are three to four times more likely to die of cancer than nonsmokers and overall, the risk to smokers is two times greater than for those who don't smoke.[11]
- More than 550,000 Americans will die of coronary heart disease this year, and up to 30 percent of those deaths will be attributable to cigarette smoking.[13]
- Heavy smokers have a 200 percent greater risk of dying from

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coronary heart disease than nonsmokers, and overall, the risk for all smokers regardless of the amount smoked, is 70 percent greater than for those who don't smoke.[13]

- Evidence demonstrates that smoking during pregnancy has a significant adverse effect upon the well being of the fetus and the health of the newborn, including causing lower birth weight infants and increasing the risk of spontaneous abortion and neonatal deaths.[9]
- Children of smoking parents have increased prevalence of respiratory symptoms and have an increased frequency of bronchitis and pneumonia early in life.[12]

Two recent studies relate smoking directly with costly health-related events, stroke and automobile accidents.

A study has concluded that smokers who quit can decrease their risk of having a stroke by more than half when compared to those who continue to smoke, thus cutting dramatically their potential health care costs.[17]

A two-year study in Worcester County, Massachusetts, comparing the motor vehicle driving records of smokers with nonsmokers found that smokers had 50 percent more accidents than nonsmokers and 46 percent more traffic violations. The study identified several reasons for the smokers' increased risk of being involved in costly accidents and violations, including

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- o smokers' more frequent use of alcohol and drugs,
- o smokers' greater risk-taking behavior, and
- o smokers' diminished attention to driving due to the distractions associated with smoking.[18]

Individual Companies Document Costs, Consequences of Smoking

While studies conducted by individual companies have varying degrees of validity, they do offer some further insights into the price businesses pay for their smoking employees.

In a study of 40,000 employees at 27 locations of the Control Data Corporation, CDC found that smokers cost the company substantially more in health related costs than nonsmokers. The study, using health data collected from 1981 to 1984, found:

- o Smokers of one pack of cigarettes per day or more generate **health claims 18 percent higher** than nonsmokers.
- o Smokers of one cigarette to one pack per day accrue **claims costs 10 percent higher** than nonsmokers.
- o Heavy smokers have **25 percent more inpatient days** than their counterparts who do not smoke.
- o Heavy smokers are **29 percent more likely to have health claims over $5,000** than those who do not smoke.[14]

One Los Angeles company estimates production losses alone at $675

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per smoker per year. Adding longer term costs such as absenteeism, premature death, and illness would raise the cost to at least $1,000 per year for each smoker.[15]

Provident Indemnity Life Insurance Company charges its smoking employees the excess rate of their insurance coverage over that of nonsmokers, an amount in the vicinity of $300 per year.[16]

Conclusion

Although estimates of the total cost of smoking to the nation in general, and to businesses in particular, differ, there seems to be no disagreement over the basic, overriding principle: Smoking is the single biggest cause of premature death and disease in our country, and U.S. businesses are paying for it heavily through increased health care costs, lost productivity, and human suffering. The implementation of policies that restrict or eliminate smoking within businesses are one inexpensive, yet effective step that companies can take to discourage smoking by their employees and to vividly illustrate their concern for health, as well as for their bottom lines.

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